

PROCEEDINGS
OF THE
ROYAL SOCIETY OF MEDICINE

EDITED BY
J. Y. W. MACALISTER
UNDER THE DIRECTION OF
THE EDITORIAL COMMITTEE

VOLUME THE NINTH

SESSION 1915-16

PART III

ODONTOLOGICAL SECTION	SECTION OF OPHTHALMOLOGY
OTOLOGICAL SECTION	PATHOLOGICAL SECTION
SECTION OF PSYCHIATRY	SURGICAL SECTION
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Odontological Section.

President—Mr. H. BALDWIN.

(November 22, 1915.)

INAUGURAL PRESIDENTIAL ADDRESS.

I THANK you, gentlemen, for the very great honour you have accorded me by placing me in this Chair. I appreciate it very much indeed, but feel much misgiving when I compare myself with former Presidents of this Section and of the old Odontological Society, their names constituting the long list of those most famous in the history of the dental profession in this kingdom. I thank you very humbly.

At the present time it is difficult to think very seriously of anything except in its relation to the War. Admirable as it was, I find it hard to emulate the spirit of Archimedes, who scorned to turn from his scientific work even in the presence of the most imminent personal danger.

The great importance of a clean, healthy and efficient mouth and teeth is now recognized by the heads of our Army Medical Department, and a great deal of dental work is being done for our troops at home and abroad. Not by any means enough, but still a great deal. At the Royal Dental Hospital thousands of soldiers of our new armies have already been treated, and the staff there has been confronted with a vast number of exceedingly bad and neglected mouths. The task of quickly restoring them to health and function has often proved an impossible one. Wholesale extraction of the septic remains of decayed teeth has been too often the only choice, leaving the mouth practically edentulous. An edentulous soldier cannot go on active service without artificial substitutes, and as the mouth requires months to settle down after numerous extractions before teeth can be permanently fitted, and then a further period of several weeks under observation for easing and adjustments of artificial teeth, in these cases they are likely to prove a complete failure. There have been hurry, scurry and makeshift

which is one of the evils accruing from having to raise an adequate number of men in scrambling haste. Under a system of universal military or national service we should have soldiers in quantity—and, let us hope, with efficient mouths as well as with arms, equipments and ammunition. An orderly and automatic system in obtaining a regular supply of recruits would, no doubt, be correlated to an equivalent provision among other things for their physical requirements.

As matters stand, there is an abnormal amount of skilled dental work required for our soldiers, both here and at the various Fronts. Added to this there is the very important field of injuries of the jaws. This requires the highest specialized skill of the dental surgeon, and can only be properly approached by the dental surgeon experienced in this particular branch of his work. His advantages in technical training and daily experience place him in unrivalled superiority in this domain. Dental work, especially in connexion with injuries of the jaws, is so important and so distinct that it deserves to be placed under the general direction of a dental surgeon of great authority and experience, and holding high rank in the Royal Army Medical Service, who should organize and conduct the dental service in the Army, and who should command the confidence of the directors of the Army Medical Service and of the dental profession. Such an officer would, I hope, be able to introduce very necessary improvements in the organization of the dental work, and important increase in the utilization of dental skill. A great many civilian dentists are necessarily, and very properly, being employed, and probably will continue to be employed, to attend to the teeth of soldiers, but their work should be inspected, or liable to be inspected, by senior and experienced dental inspectors, who, without multiplying official complications, would know how to eliminate the inefficient or dishonest practitioners. The great base hospitals for wounded soldiers have no dental surgeons appointed on their staffs. Dental surgeons are attached to them and employed there in an irresponsible sort of way, but the immense importance of their work is apparently not properly understood or officially recognized. I do not think this want of appreciation can continue to give the best results for long. Gunshot wounds of the face involving the jaws are among the most terrible of all injuries. To treat them anything like efficiently the dental surgeon is absolutely indispensable; and not merely an ordinary dental surgeon, but a dental surgeon of pre-eminent skill and experience in this class of case. His work lies largely in mitigating or preventing altogether what would otherwise often be horrible disfigure-

ment and abrogation of function. The terrible deformities which may result from inefficient treatment in cases of shattered lower jaw must be seen to be believed. The unfortunate sufferers from such injuries deserve that everything skill and experience can do shall be done for them. They should be taken in hand by the dental surgeon very soon after the receipt of the injury, and his work in re-assembling and fixing the portions of a shattered lower jaw should precede almost all other treatment; thus displacements due to muscular contraction and contractions of the soft tissues can be reduced to a minimum. None but the dental surgeon experienced in such work can see the possibilities of remedial treatment in these difficult cases. The technical work is so specialized and so elaborate that it requires a properly equipped mechanical laboratory manned by trained dental mechanics working under the orders of the dental surgeon. The laboratory should include a first class X-ray department, again operated by experts in this particular branch of work. Therefore, it is highly desirable that these cases should be collected together. Special hospitals should exist at one or more centres entirely devoted to extensive and complicated injuries of the jaws, especially the lower jaw. The hospital should have beds so that the cases could be sent there direct from the front, and undertaken by the specialists as early as possible after the injury. As quickly as possible the patients might be transformed into out-patients of the same hospital, and the beds set free for new cases. By their concentration on this class of case, the skill and knowledge of the specialists would become still more highly developed; the treatment would be improved, and the patients benefited to an extent which would not fail to be gratifying to all concerned.

The injuries we are considering are at times, perhaps, the most pathetic which can be inflicted upon the human frame, and the life-long misery entailed may be so great that their most terrible feature may indeed be that they are not necessarily fatal. We ought to ensure the best possible being done for our gallant men who fall into such evil case.

Now, to turn to the general question of the incidence of dental caries and septic infections of the alveoli, which together are the commonest of all diseases. I would like to put the question: How can we best prevent them? Can we ever return to the condition of dental healthiness and immunity from these diseases that was enjoyed by our ancestors of, say, 500 years ago? Honestly, I think we could approach it very nearly by adopting the proper procedures—at least as far as the future generations are concerned. Our knowledge of the

pathology and ætiology of dental caries is very considerable, in spite of all the reactionaries may say. Briefly, dental caries is caused by insufficient friction—by insufficient mastication of solid foods, combined with the too free use of sticky carbohydrate feeding. Again, briefly, gingivitis and chronic septic infections of the tooth-sockets, or pyorrhœa, are caused by insufficient friction, but in this case, on the gums. Friction on the gums prevents congestion. It keeps the gum tissue hard, tight, and healthy. By insufficient friction the edge of the gum is the first to suffer. It becomes congested, thickened, softened, and lowered in resistance, and therefore becomes a prey to bacterial and, perhaps, protozoal infections. When once infected, and still in the absence of proper and sufficient friction, the organisms can invade the adjacent tissues—i.e., the pericementum and alveolar bone. Result—loss of teeth by progressive ulceration of the edge of the pericementum, osteoporosis and absorption of the alveolar bone and pyorrhœa, the whole vicious process depending upon want of physiological friction. For healthy physiological friction we require active, free, and forcible mastication, postulating teeth strong and exempt from every kind of pain or tenderness. The mouth must be thoroughly functional in every part. This means a radical alteration in the kind of food given to children in the general direction of a simple and harder diet. In our highly civilized community luxury is so ingrained that we must supplement natural physiological friction by friction which is artificial. Every square inch of the gum tissue on both external and internal surfaces of the alveolar processes, and behind the backmost teeth, must be vigorously shampooed with a sufficiently stiff tooth-brush twice a day. This will often cause slight bleeding, even when the gum is in good condition; and for this reason, and also for keeping the brush clean and sterile, an antiseptic must be used during the brushing. Common salt is a suitable antiseptic. The great public should be taught to brush its gums with salt. There seems to be no better antiseptic for the mouth than chloride of sodium.

The prophylactic effect of regular daily and sufficient brushing of the gums is quite astounding. Whether it acts by stimulating the circulation of blood or circulation of lymph in the tissue, or by stimulating the living cells and fibres of the tissues, or by removing or preventing the formation of a bacteria-laden coating of mucus and dead and infected epithelial cells, or whether by bursting and destroying small vessels which are becoming swollen and threatening congestion, it is not possible to say, but probably by a combination of

all these. It certainly prevents the formation of subgingival calculus, partly by not allowing a loose exaggerated free edge to the gum to exist, and partly by preventing a morbid oozing from the gum, whether sanious, serous, or purulent, any of which kinds of exudation when mixed with saliva actually produce particular kinds of calculus in this particular situation. The net result of the regular and sufficient friction in preventing gingivitis and its severe sequelæ, and also in preventing interdental caries, is exceedingly important. Its general adoption would prove of the greatest possible service in acquiring immunity from both dental and gingival diseases.

The gospel of hard feeding and gum-brushing has still to win its way. The parents and school-teachers must be converted, and the children must be trained to do the right things from earliest years—i.e., as soon as they have teeth. Regular visits to the dentist should be made, and, if any caries appears, it should be immediately stopped, especially any incipient crown cavities in six-year molars should be filled at once; and so tenderness and pain, chief hindrances to forcible mastication, will be prevented. The children must be taught the hygiene of the mouth—the most important chapter of which is gum-brushing. Thus it is obvious that it is school dentistry in the elementary schools to which we must look for the acquirement of something like immunity for the future. The influence of the dentist in inculcating the doctrines of dental hygiene will be as valuable as his actual operative ministrations. At this age the individual can be given a fair start, and can receive the necessary knowledge and instruction to keep on a high level of dental health and dental efficiency. A few but regular visits to the dentist at the school clinic would start the child upon a course which would easily suffice to prevent any serious trouble, and would enable the average individual to go through life without loss of permanent teeth and without pathological septicity in any part of the mouth.

What a vista of increased comfort, health, efficiency of the mouth, and therefore of the whole body, is here presented! And what a small expenditure of time, money, and trouble is demanded in anticipation of such a great and far-reaching reform!

It has lately been said the British soldier is in reality the product of the elementary school. Let it be to the credit of the elementary school in the future that the product goes forth with a clean and healthy mouth, and with the knowledge and the resolution necessary to keep it so throughout life.

(November 22, 1915.)

Comparative Studies in Calcification.

By J. HOWARD MUMMERY, M.R.C.S.

I HAVE entitled the present communication "Comparative Studies in Calcification" as I wish to draw attention to certain physical phenomena and to the process as it occurs in crustacea, mollusca, and brachiopoda which, I think, throw some light on the subject. This is chiefly the record of a preliminary study which was undertaken in preparing the two papers on calcification which have already appeared,¹ and which is only shortly referred to in those papers. The references to the published portion will be chiefly by lantern slides and microscopical preparations, which will, I think, demonstrate the points I wish to emphasize more fully than a description by words only.

At the soirées which were held to commemorate the opening of this new building, Mr. Deane Butcher gave a very interesting demonstration of Professor Leduc's methods of producing osmotic growths in colloidal solutions [5]. Wishing to study these phenomena under the microscope, I experimented with various substances. I was most successful with crystals of sulphate of copper in a solution of silicate of sodium. When particles of copper sulphate are dropped into a weak solution of the silicate (about 5 per cent.) on a microscope slide, an immediate production of small tubes takes place from the finer particles, and from the larger masses of crystals a membrane is protruded, forming a small globular eminence. A succession of these is formed by a series of small explosive impulses, and the membranes then grow out steadily into a tube which is seen to pass rapidly across the field of the microscope, often attaining the length of an inch or more, and terminating in rounded and closed extremities. This is an osmotic growth of the same nature as those described by Professor Leduc, and produced by him in various substances with many different salts. The growth of these tubes is due to the rapid passing of water through the membranous wall from the

¹ J. H. Mummery, "On the Process of Calcification in Enamel and Dentine," *Philosoph. Trans.*, B. ccv, pp. 95-114; "On the Nature of the Tubes in Marsupial Enamel and its Bearing upon Enamel Development," *ibid.*, pp. 295-314.

surrounding solution by osmosis, the distension by the water pushing the tube forward, the resistance of the membrane at the same time preventing the lateral bulging of the tube.

These particular points, however, have not such an important bearing on the present investigation as the following: As the tube nears the end of its growth, in some places lines are seen to be drawn across it, dividing the whole tube with a distinct septum.

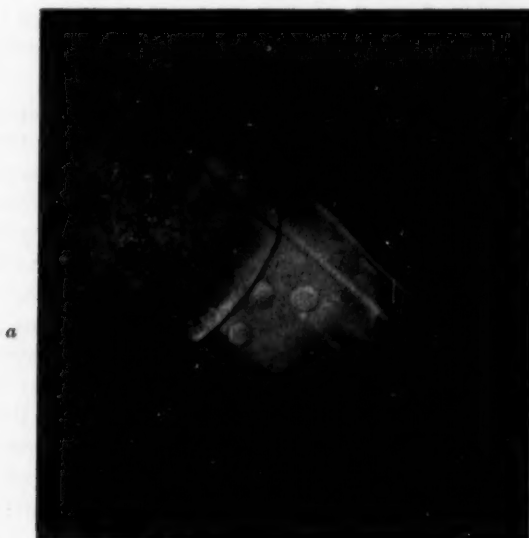


FIG. 1.

Osmotic growth formed by copper sulphate in silicate of potash. Transverse membrane formed opposite *a*, showing crystalline forms above it and calcospherites beneath.

These are Traube's membranes of precipitation, but it is difficult to explain what determines the production of these membranes in this position as they are usually only seen in a few tubes in one preparation.

Several eminent physical chemists to whom I have submitted this problem are unable to explain it. It can hardly be due to fractures, as has been suggested, for the course of the tube is not altered as one would expect to see if this were the cause. After some time, varying from half an hour to an hour, small blue spherical bodies appear within

the tube upon the copper sulphate side of the membrane, the tube on the other side remaining quite clear. These little bodies soon lose their smooth outline and have a radiating appearance, as if formed of little bundles of acicular crystals united at the centre, and many of them break up into small shining particles. In some slides which had not been allowed to dry, but had been kept under observation for some days, I found these small crystals piled up on the membrane on the copper sulphate side, and on the other, or distal side, several large colourless spherical bodies lying beneath the membrane. These bodies show under high magnification both radial and concentric lines, and react to polarized light exactly in the same manner as do the similar calcospherites produced in gum or albumen.

Dr. Lovatt-Evans, who kindly repeated the copper sulphate experiment for me, points out that the little bodies above the membrane, which are first rounded and afterwards acicular, may consist either of a silicate of copper or of a double salt of lime and copper, as their spherical form on their first formation would suggest. The calcospherites beneath the membrane are probably composed of silicate of lime. The lime salts are probably derived from the tap-water in which the silicate is dissolved. The interesting point is that true calcospherites are formed by dialysis through membranes which are formed in inorganic solutions by purely physical processes.¹

It is unnecessary to remind you that Graham, as the result of his experiments on the diffusion of liquids through membranes, divided substances into crystalloids and colloids, crystalloids having the power when in solution of passing easily through membranes; colloids, on the other hand, passing through with difficulty or not at all. The particles of a colloid solution are held together by a very feeble force; they consist of very fine particles in a state of suspension, rather than solution, in the solvent. The exact line of demarcation, however, between crystalloids and colloids is not always very pronounced, and according to Krause there is a steady transition from the crystalloids to the colloids. As Loeb [6] says:—

“Living organisms may be considered as chemical machines, consisting essentially of colloidal material, which possess the peculiarities of automatically developing, preserving, and reproducing themselves.”

He also says: “Living organisms are built up of substances which

¹ The small bodies above the membrane form also when distilled water is used to dissolve the silicate, but, as Dr. Evans says, this would not be a proof of the absence of lime, as calcium salts are present in ordinary specimens of sodium silicate.

are essentially colloidal in character, which may be either in the liquid state ('sols') or in the solid state, when they are known as 'gels.'

"The proteids, certain carbohydrates, like starch and glycogen, and higher fats belong to the colloids, while their products of cleavage, as dextrose, may belong to the crystalloids.

"Life depends upon the existence of these colloidal solutions in the cells. All organic agencies which bring about a general gelation, bring life to a standstill, and such standstill is permanent in case irreversible 'gels' are formed, such as originate if proteins are heated. The liquid proteins of our body coagulate at a comparatively low temperature, and this is the reason that at a temperature of about 45° C. the cells of our body die very rapidly.

"There are, however, conditions in which the transformation of 'sols' into 'gels' does not lead to death, but the formation of important morphological structures—Traube's [13] membranes of precipitation—for investigation showed that when two liquid colloids come into contact (or a colloid and a crystalloid) solid membranes may be formed."

Traube "investigated the formation of the membranous plant cells, and was led to the conclusion that the formation of these membranes and the peculiarity of the cell to grow depends upon a simple physical process."

"Certain colloids form a precipitate where they come in contact with each other, and this precipitate is impermeable for either colloid. The precipitate must therefore assume the shape of a thin film, which prevents the further action of the two colloids on each other." This film Traube called "the membrane of precipitation." The cell membrane (or rather the surface film of protoplasm) makes a diminutive chemical factory of the contents of this cell by shutting it off from its surroundings, and enables each cell to lead a specifically different life from the neighbouring cells. "Traube thus became the originator of the modern theory of the growth of cells, which assumes that the growth is caused by the cell absorbing water in consequence of its osmotic pressure being higher than that of the surrounding solution."

It has been generally considered that these membranes of precipitation are permeable to water only, and not to salts, while others say they do not allow sugars or salts to pass through at all, or not equally well; but at all events, in organic tissues, permeability to salts is evidenced by the absorption of salts by the cells of plants, as has been proved by many experiments.

Dr. Lovatt-Evans suggests that in the experiments with the copper

sulphate we are dealing with a membrane which is only to a limited extent semi-permeable (by semi-permeability is meant that the membrane is permeable to water only), and I think this is evident from the appearance of these calcospherites.

Loeb is of opinion that "cell walls are not impermeable to salts, and that there is only a difference in the rate of diffusion of the various substances, many salts diffusing only very slowly into the protoplasm." We thus see that under purely physical conditions we have a dialysis of salts of lime through these inorganic membranes, and this observation points to the suggestion that the same thing may occur from physical causes in the calcification of the various hard tissues of man and animals.

The existence of membranes which act as dialysing membranes in the enamel organ has been denied by some. Leon Williams is, however, convinced of their existence, and my own preparations, I think, make it very evident that such membranes do exist. The outer ameloblastic membrane of Williams separates the cells of the stratum intermedium from those of the ameloblasts, and the inner ameloblastic membrane divides the ameloblasts from the forming enamel. These membranes are very clearly visible in the photograph of the tooth germ of the kitten, where an active deposit of enamel is proceeding. Before the commencement of calcification the cells of the stratum intermedium are not shut off from the ameloblasts, and the cells of this layer may be seen in places lying between the ameloblast cells. But after calcification has commenced the stratum intermedium is distinctly and sharply separated from the ameloblasts by a limiting membrane. That this membrane really exists is, I think, evidenced by the photograph, which shows the cells of the stratum intermedium in a regular row above the membrane, while beneath it the ameloblasts have fallen away, leaving a distinct line of tissue running parallel to the cells of the stratum intermedium.

In the dentine, as was pointed out by Höhl, the odontogenetic zone between the odontoblasts and the forming dentine shows the tubes passing across it occupied by the dentinal fibril, but when the section has been stained with silver nitrate, the sheath of Neumann is not visible in this zone, although clearly defined in the calcified dentine. Höhl concluded that this sheath is in some way connected with the calcifying process, and I have ventured on the suggestion that the sheath appearing as calcification commences is really a dialysing membrane, the salts from the odontoblast and their processes, passing through this

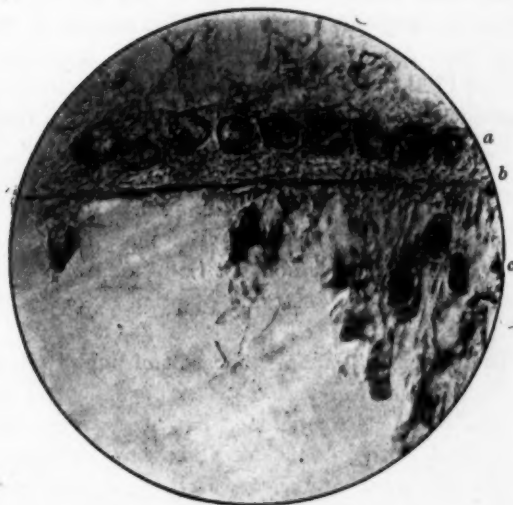


FIG. 2.

From the enamel organ of *Macropus*. a, Stratum intermedium; b, outer ameloblastic membrane; c, ameloblasts detached from membrane in one part. ($\times 1,000$.)

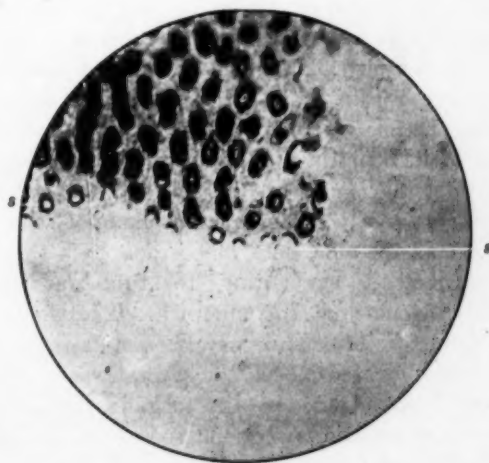


FIG. 3.

Human dentine stained silver nitrate (pyridin process), showing sheaths of Neumann (s). ($\times 800$.)

and combining with other constituents of the hard substance of the dentine within the organic matrix. The existence of the sheath of Neumann has been denied by many observers, and most recently by Professor Römer [11], who states that in his silver nitrate preparations, when the stained fibril had fallen out of the dentine, only a hole was left in the matrix with unstained margins. I think, however, his preparations could not have been fully stained, as in those I made with the Ramon y Cajal silver method the empty tubes are surrounded with a very distinct black line, and at the margins of the section segments of the tubes are seen which show the membrane very distinctly. The sheath can also be demonstrated by other stains, such as toluidine blue.

How far these membranes are the result of purely physical agencies, and to what degree they are affected by physiological and vital conditions, we are unable to conclude, but it is seen that very firm membranes do arise in the absence of all organic substances. There is, however, a tendency when any theory is brought into fresh prominence to endeavour to interpret phenomena exclusively on this view, but this is an error we should especially seek to avoid. In such a complex structure as the animal body many agencies are at work—chemical, physical, and vital—and they have all to be taken into consideration in drawing any conclusions as to the processes which take place in the living organism. As Professor Philip [8] says: "A purely physical theory of the exchanges which take place across a living membrane is inadequate; there is a physiological as well as a physical permeability."

The tube which I pass round for examination shows an artificial shell, illustrating the formation of a calcified body of calcium phosphate by the diffusion of its constituent salts through a colloid. A fragment of calcium chloride was placed at one side of the bottom of the glass tube and a few crystals of the rhombic phosphate of soda ($\text{Na}_2\text{HPO}_4 + 12\text{H}_2\text{O}$) on the opposite side, held in position by a thin layer of firm gelatine poured in as it was setting; the tube was then filled with gelatine mixed with one-third egg albumen. The whole mass became solid in a few minutes. After an interval of ten days the shell-like body began to form and increased in size for several weeks. Small star-like particles appeared suspended over the forming shell, which gradually coalesced into the completed formation; somewhat similar experiments were made by Professor Harting and Dr. Ord.

THE ARTIFICIAL PRODUCTION OF CALCOSPHERITES.

In the year 1858, Rainey published his work on molecular coalescence [10], showing that lime salts were deposited in colloidal substances not in a crystalline, but in a globular form, the globules having a very definite structure and arrangement. He also showed that in many animal organisms, as mollusca and crustacea, similar forms appeared during the process of calcification of the shell. The first experiments were made with a solution of carbonate of potash in gum (which contains salts of lime). The bodies so produced were globular in form, showing concentric lamination and radial striæ. Professor Harting, of Utrecht [4], independently carried out a similar research and succeeded in producing a great variety of these spherical bodies in various substances, his most instructive experiment being one in which small fragments of calcium chloride and of sodium bicarbonate were placed on the opposite sides of a flat dish containing egg albumen. The dish was left perfectly still and undisturbed for two or three weeks—the salts diffusing through the albumen from the opposite sides formed a crust upon its surface. This crust was found to consist of a deposit of carbonate of lime in the globular form, showing a great variety of spheres and disks of varying size, and very similar to those produced in gum in Rainey's experiments. He found that they retained their form after they had been subjected to the action of an acid, some portion of the lime having become so intimately blended with the albumen that it remained in combination with it. To this substance, left after decalcification, he gave the name of "calcoglobulin," distinguishing the calcified globular bodies by the name of "calcospherites."

If a microscopic slide be prepared with gum or albumen containing carbonate of potash or soda and examined under the microscope, minute granules appear after a short time, which increase in size by coalescence until small globular bodies are formed—the process of molecular coalescence described by Rainey. Both Rainey and Harting found that when calcium phosphate was present in the solutions in excess of the carbonate, globular bodies were not formed, but the deposit was crystalline, but if there was only a small proportion of phosphate to carbonate, larger and more perfect spheres were produced than with carbonate alone. This observation has a very important bearing on the subject of the present paper, as in completed dentine and enamel the phosphates are largely in excess.

In repeating Professor Harting's experiment with albumen I placed on one side of the dish, equidistant from the calcium chloride and the sodium carbonate, a few fragments of sodium phosphate ($\text{Na}_2\text{HPO}_4 + 12\text{H}_2\text{O}$). Examination after the lapse of three weeks showed that in the neighbourhood of the sodium phosphate the crust formed on the surface of the albumen was entirely crystalline. Nearer to the calcium chloride, however, globular bodies had appeared, but they were not perfect in form, and some globules had acicular crystals projecting from them in many places. Further away from the phosphate very large and perfect calcospherites were found. In dealing with calcification in dentine and enamel in a former paper I referred to this and to the probable explanation of the fact that calcospherites are deposited in both these tissues, although analyses of calcified material show a very large preponderance of phosphate.

In the works of Harting and Rainey a full and minute description is given of the various forms in which the calcific material is deposited, and it would be superfluous to describe them here, but the illustrations in the book of drawings on the table show a great variety of forms obtained in my own experiments. Two main forms of calcospherites may conveniently be distinguished—those in which radial striæ are most evident and those specially marked by concentric lamellæ or rings, although there are many which show both structures. In many parts of the calcareous crust in the albumen experiments the globules are seen to have coalesced, they are often arranged in rows around a central extension filled with minute spherites, and in other places where in contact they have become fused into larger bodies, hour-glass-shaped, and double spherites being very frequent. In some parts there is an accumulation of the globules into a mass like a collection of cells, where they are seen to have assumed an hexagonal form from mutual apposition. Several have a rosette form bearing a strong resemblance to the corolla of a composite flower. These seem to be chiefly the larger ones, which are showing a tendency to disintegrate, the central part breaking down into the smaller elements. This is especially well seen in the similar bodies found in the carapace of the prawn, referred to later. In many of the larger radial spherites, beside the delicate radial striæ, wider spaces are seen extending from the centre to the circumference, of an irregular shape, and evidently indicating a splitting or dividing of the spherite into sections. This is frequently seen in the disintegrating globules in enamel, presently referred to.

CALCOSPHERITES IN THE LIVING ORGANISM.

These were examined in the shell of the crab (*Cancer pagurus*), in the Norwegian lobster (*Nephrops norvegicus*), and in the prawn (*Palæmon serratus*). In the prawn, as pointed out by Rainey, the deposit can be very easily studied, and in many of my preparations of portions of the carapace mounted in Farrant's solution the calcospherites are shown in all stages of coalescence and disintegration. In the prawn, where the deposit takes place in a membrane which lines the under surface of the shell, spherites of very large size and perfect form are to be seen, showing the same shapes and internal structure as in those formed artificially, but larger and more perfect. Rosette and elongated forms are seen, which on comparison with the artificial ones are seen to be of the same type; in many places they are breaking down, some show radial splitting, others are breaking up into smaller elements, and in the large masses of coalesced calcospherites this disintegration at the centre of the accumulated bodies is very noticeable. The slide shows a ground section of the margin of the shell of the crab, in which the scattered spherites in the membranous layer are well shown and also the manner in which they coalesce and become incorporated in the forming shell. In the part figured there are no tubes, although true tubes are found in many parts, but in this situation (the shell near the base of the claw) radiating and horizontal lines are very evident, formed by the structural elements of the accumulated calcospherites. A photograph taken by dark-ground illumination shows very clearly that the transverse lamination is due to the consolidated calcospherites, the lines being seen in the separate bodies as well as in the formed shell.

Rainey, in endeavouring to push the analogy between the formation of the crab-shell and human dentine, went so far as to deny the existence of the dentinal tubes as such, and compared them with the longitudinal striæ in the crab-shell continuous with those in the separated spherites. While in the crab the small vertical wavy lines certainly appear to be due to the radial markings in the globular bodies, there are no radial markings in the spherites found in dentine, and therefore no vertical markings due to these bodies. The true tubes in the crab-shell are more scattered than these little vertical lines, which, as Rainey says, near the forming edge appear as mere lines of dots, as they do also in the isolated spherites. When this author made his investigations, the true nature of the dentinal tubules and their contents was not generally known, and in consequence the part of his work

which deals with the analogies of structure in teeth and bone is of little value. The cell theory in his day was also in its infancy, and in his work, while he shows the very large part which physical processes play in the living body, he scarcely sufficiently acknowledges the influence of the vital conditions with which these processes are associated. In the small Norwegian lobster which I examined for this purpose, a ground section of the claw shows the same fibrillar basis substance in which the calcospherites lie, but in this creature they were, in this situation, chiefly of one form, resembling the flower of a pansy, and it can be seen that the separate portions of these bodies are scattered in the forming shell, like separated petals of the perfect flower. More towards the centre, away from the calcifying margin, it can be seen that the shell is finally built up by the disintegrated particles of the original spherites.

I think it is evident in all these examples that the shell is ultimately formed by the disintegration of the calcospherites, which may take place either previous to their incorporation with its substance or after that incorporation has taken place.

In all these organisms which I have examined, in crustacea, mollusca and brachiopoda, there is, as I have already pointed out, a distinct, finely fibrillar basis substance which appears to serve as a support to the material, secreted by the animal, in which calcification takes place. This fibrillar substance is of a clear, apparently structureless, homogeneous nature, and does not appear in any way to determine the actual arrangement of the calcareous particles, but simply seems to serve as a binding material. It is not a connective tissue and shows no connexion with cells.

The view held by Bowerbank [2] and Carpenter [3] with regard to the formation of shell in the mollusca was that "shell is an organic formation growing by interstitial deposit in the same manner as in the teeth and bones of the higher animals." This view, which accorded with the opinion held at the time, that enamel and dentine were formed by actual deposit in the formative cells, supposed that the same thing occurred in the mollusca, and that in "pinna" and in other shells the membrane left after decalcification represented the organic cells in which the deposit of the lime salts had taken place.

The view now generally held with regard to shell formation is that it is due to a process of secretion. This is more in accordance with modern views as to calcification in the teeth, which is considered to be produced by deposit in a material separated and secreted by the cells,

and not by direct conversion of the cell into the calcified substance by interstitial deposit. The organic remainder after decalcification of a shell of pinna would not then indicate that these hexagonal outlines represented cells, but were due to the remains of the fibrillar material present in all shells, in which the deposit takes place—the remains of the basis substance between the crystalline calcareous plates by which the prisms of this shell are built up. In the brachiopod terebratula, the fibrillar substance is very clearly to be seen in the mantle, and also in the remains of the decalcified shell there is no evidence of any deposit in cells but in a very strongly marked irregular network of clear homogeneous material.

Dr. Carpenter in his work on the microscope, published subsequently to his original paper, is disposed to agree with Professor Huxley "in the belief that the entire thickness of the shell is formed as an excretion from the surface of the epidermis," and that the periostracum is "converted into the likeness of a cellular membrane by the pressure of the prisms that are formed by crystallization at regular distances in the midst of it."

In the shell of terebratula and allied species of brachiopoda some very interesting points are to be observed which appear to have a strong bearing upon calcification, and would seem to serve as an illustration of the process occurring in the dentine. The brachiopoda were at one time classed with the mollusca, but are now separated into a distinct group. As is well known, the shells of the Terebratulidæ are penetrated by numerous tubes which enter the shell at the mantle surface and terminate beneath the periostracum in expanded ends. These tubes traverse the layers of the shell, which are of a prismatic structure, at right angles or nearly so. They are filled with a soft substance containing an abundance of cell-like bodies and granules. These cellular contents of the tubes can be traced back to the mantle, where they have a definite confining membrane and are seen to take their origin from large bulb-like bodies lying in the fibrillar mantle substance and arranged in rows. In ground sections of the calcified shell from specimens of *T. vitrea* which had been placed in spirit in the living condition, the cellular elements in the tubes are very conspicuous and take the stain easily; unstained granules can also be seen. The bulb-like bodies, which lie in a very clearly defined fibrillar matrix, have a peculiar structure. The somewhat flattened and expanded or trumpet-shaped end extends beyond the cell contents, and appears to be closed by a very delicate membrane, the part between the cell

contents and the terminal membrane being filled more or less with refractile granules, which are also seen between the cells. The resemblance to dentine in transverse sections of the shell is very marked, the cells in cross-section occupying the place of the dentinal fibrils and lying within a circular tube. In longitudinal section the tubes show alternate dilatations and contractions in the different layers of the shell. In the layer of the shell where the tubes attain the greatest dimensions they measure $16\ \mu$ in diameter, the bulbs averaging $20\ \mu$ in width. In the partially decalcified shell these bulb-like bodies can be seen attached to the inner margin of the shell and hanging out from the tubes by their processes. In transverse section many of the tubes are seen to have short radial branches, which appear straight and needle-like. These were first described in terebratula by Professor Quekett. Transverse sections of the calcified shell also seemed to show a definite lining to the tube walls. In order to determine this point I treated a portion of shell by the Cajal silver nitrate process, and a very definite dark line was seen around the inner margin of the empty tube. The shell was then decalcified, the lime salts being removed until only the organic fibrillar matrix was left, but the stained inner margin of the tube was not affected by the acid, and remained as a stained black ring in the organic matrix. It is thus evident that in terebratula as in dentine there is a definite lining to the tube distinct from the rest of the calcified shell, and showing the same resistance to the action of acids exhibited by Neumann's sheath in the dentine. The shell of another brachiopod, crania, is also penetrated by canals, but they break up into a number of branching tubules beneath the outer surface of the shell, and the branches, like the tubes are filled with a protoplasmic material "which has its origin in the epithelial cells which lie in the tubules." I have not been able to examine this condition, as I was unable to obtain any specimens of crania.

The existence of these highly developed fibrils in terebratula and crania has been looked upon as a very puzzling problem. A. Shipley [12], who has made a special study of the Brachiopoda, says: "By some they have been regarded as respiratory organs, but it would seem more reasonable to suppose they serve as organs to supply nourishment, &c., to the organic matrix of the shell." Professor Owen [7] suggested that these caecal processes with their continuations into the tubes of the shell might perform an excretory function, and, as he says, "be associated in that action with the depurative respiratory office



FIG. 4.

Terebratula vitrea. The bulb-like terminations of the tubes in the mantle (so-called caecal bodies).



FIG. 5.

Terebratula vitrea. The caecal bodies and tubes hanging from the tubules of the shell.

of the mantle, the probable condition of their development being the low grade of the proper branchial organization." Dr. Carpenter [3], believing that certain corpuscles which he observed in the cæcal processes were blood corpuscles, deemed it not improbable that the apparatus in question is branchial in its nature. It is not easy to understand this view, as the cæcal appendages are closed bulbs and have no direct connexion with the sinuses of the mantle containing the blood. The cells are also very closely packed together and do not resemble blood corpuscles in a plasma. With regard to the suggestion that they serve to convey nourishment, &c., to the organic matrix of the shell, Dr. Carpenter has pointed out that there is very little organic matter in the shell of terebratula, and the great abundance of these cellular elements in the tubes would seem to indicate some further function. Mr. Shipley seems to have also had some doubts on this point, for he speaks of nourishment, &c. If in the "et cetera" he includes lime salts, I think we should arrive at a more reasonable explanation. It is possible that these cellular elements may be the actual agents in the secretion of the lime salts which, by dialysis through the walls of the tubes, constitute the calcified matter of the shell. This would also be borne out by the observation which I have recorded above, that refractile granules are seen within the bulbs of the mantle, which closely resemble the granules seen in other substances undergoing calcification. Many of these granules are seen closely applied to the membrane which closes the flattened end of the bulb, and would suggest that the salts are dialysed from the body fluids in the mantle in which the bulbs lie to the interior of the bulb, and thus to the tubes of the shell.

The permeation of the shell in terebratula and crania by tubes containing protoplasmic material, the existence of a sheath similar, apparently, to that in the dentinal tubes, the presence of granules and the peculiar formation of the bulbs themselves, are certainly highly suggestive of some calcifying function, but beyond this one cannot go, as it is, of course, impossible to make any definite statements on the question, which must remain a somewhat problematical one.¹

¹ The specimens of Brachiopoda were kindly procured for me from the Naples Aquarium by Dr. Guerini.

CALCIFICATION IN THE DENTINE.

With regard to dentine, it is well known that it is built up of rounded bodies or calcospherites, as is evidenced by the contour at the margin of the odontogenetic zone in growing teeth, and also by the rounded bodies visible in the interglobular spaces. These appear to be clear and structureless, both in growing teeth and in these imperfectly calcified areas, and evidently indicate that the whole of the



FIG. 6.

The concentric calcospherites in human dentine and parallel lamination. Silver nitrate (Cajal) preparation. ($\times 800$)

calcified part of the dentine is built up by these bodies. At the borders of carious cavities, where the acid of decay has acted upon the dentine in advance of the invading micro-organisms, a fine striation is sometimes visible. Many years ago I prepared a lantern slide from such a preparation, but could not understand the cause of this delicate striation, nor could anyone to whom I showed it explain its meaning. In young erupting human teeth prepared by the Cajal silver nitrate process,

however, I found the explanation of this appearance. The striation is due to the lamination of the calcospherites, which is very evident in these sections and exactly corresponds to it both in appearance and in dimensions. The small clear calcospherites seen at the growing border evidently coalesce deeper in the dentine at this stage of development, and exhibit the concentric striæ. The appearance of concentric striæ becomes concealed by subsequent dense calcification, and is again revealed by the process of decalcification by the acids of caries—reversing, in a sense, the order of development. It is thus seen that at a certain stage in the calcification of the dentine the whole substance is made up of concentric spherites of very different sizes, which from some obscure cause get drawn out into the laminæ of which they are severally built up. It is difficult to say what determines this spreading out of the calcospherites; possibly it may be due to pressure of the consolidating tissue. The whole appearance, however, points to the conclusion that the calcifying margin at the odontogenetic border does not show the final consolidation of the dentine which goes on later within its substance. There is some evidence to show that a similar consolidation within its substance also occurs in enamel. As in enamel I think the method of calcification of the dentine illustrates the process of coalescence and disintegration of the spherites referred to by Rainey. It will be noticed that the calcospherites in dentine show concentric markings *only*, while those of enamel show only a radial structure and break up in a direction from the centre to the circumference.

CALCIFICATION IN ENAMEL.

The researches of Leon Williams [15] on enamel, demonstrated, I think beyond all controversy, that perfect fully formed enamel is a dense substance in which no spaces or uncalcified matter can be detected. The photograph, which I made from a Weil preparation of my own, was from very thin enamel magnified 800 times. It shows the transverse striation and the building up of the enamel in blocks very clearly, and also the cytoplasmic threads within the prisms. Williams believed that the cytoplasmic threads which form the Tomes's processes, and are the continuations of those seen within the body of the ameloblast cell, are consolidated by the calcification of the lime salts around them in the form of regular globules (or rather blocks) of the calcified substance, and, contrary to the opinions of Charles Tomes, Walkhoff and others, he held that the interprismatic substance is calcified separately from the

rods or prisms, although when completed so fully calcified as to form a compact tissue of rods and interprismatic substance. He assumed that the clear globules seen in the ameloblasts are the calcifying substance which is dialysed through the inner ameloblastic membrane which separates the cells from the forming enamel and is redeposited within the prisms in a perfectly regular arrangement of blocks, their individuality being marked by the transverse striation which indicates their mode of deposition. These blocks are shown to be made up of fine granules. He considered that the calcification of the interprismatic substance took place in a product of the ameloblast cells which flowed round or enveloped the calcifying prisms.

This mode of formation of the enamel is, I cannot but think, the true explanation of the process, and his photographs and preparations are very convincing. My own work on enamel, I think, fully confirms his views, as I was able to show the actual calcified and calcifying bodies within the interprismatic substance—large calcospherites being evident in this effused material from the cells, and these calcospherites exhibiting all stages of coalescence and disintegration. This was best demonstrated in marsupial enamel, where a very large amount of the basis substance is laid down before the enamel is at all fully consolidated. In the photograph on the screen, these large spherites are clearly seen and the small granules into which they break up, and with them are seen the blocks which build up the enamel prisms. This photograph, I think, shows the whole process very clearly in one field of the microscope. It would appear that in the effused material between the prisms as in artificial experiments, small granules appear, that these coalesce and form the larger bodies, which in turn again disintegrate and form the solid material which binds the prisms together.

In order to try to detect the actual manner in which the prisms were bound together by the interprismatic material, I scraped a dried developing tooth of the kangaroo taken from the crypt over a drop of glycerine on a glass slide—a small rough enamel cap only being formed. On touching these pieces with needles under the microscope, they broke up into laminæ, all in one direction, and, by repeated teasing with needles, into very fine and transparent layers, which could be examined with the high powers of a microscope. These layers lay over one another exactly like the leaves of a book, and were crossed by a very fine fibrous striation. The lines of forming enamel rods are clearly seen, and lying between the laminæ are rounded bodies which a higher magnification shows to be true radial calcospherites. Some of these were splitting up along the

lines of the radii, and many were separated into four portions. Lying along the edges of the forming prisms were numerous rounded bodies of various diameters, and small granules are seen distributed over the surface. I think, therefore, that these preparations indicate that the enamel is formed in laminae which lie vertically to the upper surface of the enamel, that the calcification takes place by the deposition of small granules as in artificial experiments, that these coalesce and form the large radial calcospherites, which, as calcification advances, are again disintegrated and form a very minutely granular or often quite clear substance which cements the columns of the prisms together. In human enamel, which is more rapidly calcified and is a denser material than marsupial enamel, these large bodies can also be seen, but I had to search a great many preparations before I was able to see it. The photograph shows calcospherites in the laminae of developing human enamel examined in the same way as the marsupial material. One very curious observation was that the forms of the calcospherites in human and marsupial enamel were different. In the marsupial enamel the spherites were true spheres, in human enamel the greater number are oval, as is shown in the mass of calcospherites lying just beneath the enamel organ on the surface of the developing molar of a child aged 6 months.

Mr. Douglas Caush many years ago showed that stains entered the enamel and sometimes obtained a diffuse staining in places. In grinding down sections of young teeth treated by the fuchsin method, I have often found deeply stained laminae that had to be ground away to obtain a clear section of the enamel. These are, I believe, the laminae stained by Mr. Caush, and which I have described in the teased specimens of marsupial enamel. In the adult tooth they eventually become involved in, and concealed by, the dense calcification.

In a recent paper Professor Walkhoff [14] maintains that while the enamel of unerupted teeth remains for a long time under the influence of the enamel cells, after eruption it is a dead tissue which cannot obtain lime salts either from the dentine or from the saliva. There seem, however, to be many appearances in enamel which do not support this contention. The enamel of the young kangaroo shows the rows of globular deposits of lime salts in the prisms in the erupted tooth. Pickerill, speaking of penetration from without, showed that the portion of enamel in young teeth which is first formed is not permeable to stains, but the last-formed portions are permeable, and specimens which I prepared by the Weil process after impregnation with silver



FIG. 7.

Fragment of enamel of *Macropus rufus* teased out in glycerine, showing arrangement of enamel in laminae and calcospherites lying within them.

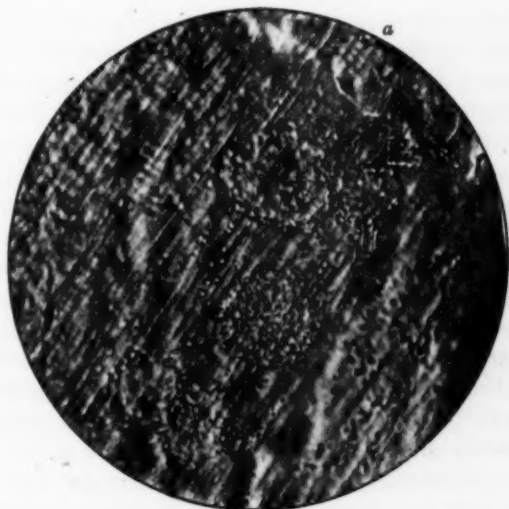


FIG. 8.

Portion of enamel from specimen shown in fig. 7 more highly magnified, showing the beading of the prisms (a) and large radial calcospherites and granules in the laminae. ($\times 1,000$.)

nitrate show this fact very clearly. A penetration of the enamel by the dentinal tubes is also easily demonstrated, and by the method of injection of alcoholic fuchsin from the pulp cavity, as described by Dr. von Beust, it is seen that not only the club-shaped bodies in which some of the tubes terminate are stained within the enamel, but also fine terminations of the tubes passing a considerable distance into its substance. Professor Walkhoff also considers that the cement substance described by von Ebner and by other authors as the interprismatic substance does not exist, and is in reality the outer or cortical layer of the prisms, or, as he prefers to call them, the "Zentralkörper." The cement substance, however, is very evident in the teeth of marsupials, both in longitudinal and transverse sections, and I think we may look upon marsupial enamel as giving us the key to the formation of other mammalian enamels.

In marsupial enamel, as I have shown above, calcification takes place in the laminae between the prisms, and I think it is very evident that these large calcospherites are produced quite independently of the prisms upon which they lie—the prisms are calcified in rows of minute spherical bodies, which appear to be formed within the Tomes's processes of the ameloblast, and are the first part of the enamel to receive the calcific deposit, while the interprismatic substance is formed subsequently in the organic fibrillar matrix, which is seen throughout the enamel in the kangaroo. The calcification in marsupial enamel does not, however, proceed to complete obliteration of the cement substance, much of which remains uncalcified, as the alcoholic fuchsin method clearly shows. This interprismatic substance is very visible in the tooth of the kangaroo.

I certainly cannot reconcile the appearances in my preparations with Professor Walkhoff's view that the whole calcification of the enamel takes place centrifugally from the prisms. When human teeth are decalcified by acids, either artificially or by caries, we know that the prisms become separated from one another, apparently from the decalcifying acid first acting upon the interprismatic substance; this would not indicate that it was more fully impregnated with the lime salts, but that they are held together between the prisms in a less compact condition and sooner yield to decalcification.

The enamel of the rat, in which the fibrillar structure is also very marked, gives strong evidence of a very considerable amount of cement substance between the prisms. It would appear that in less perfectly formed enamels a cement substance is very evident, and that the denser and more completely formed the enamel the less is this substance in evidence, its existence being veiled by calcification, so that in the

enamel of the primates the evidence of its presence in the perfectly formed portions of the tissue is very slight or altogether absent.

Professor Walkhoff finds in the erupted teeth of the anthropoid apes areas of imperfect calcification similar to those found in human enamel, and apparently considers that as no after-changes take place in these areas the enamel is a dead tissue. I do not think it necessarily follows, as when these defects occur from some passing arrest of calcification during the development of the teeth they are closed round with perfectly formed tissue, and would remain as imperfections, exactly as do the interglobular areas in dentine, in which no further changes take place. This, however, would be no argument against the last-formed normal layers of enamel being still when erupted not fully calcified, and a dialysis of lime salts from the saliva may take place as suggested by Pickerill, through the covering membrane of Nasmyth, which is all that remains of the enamel organ, so that while quite agreeing with Professor Walkhoff that no change takes place in these areas of incomplete calcification, I think there is strong evidence that a further consolidation of the enamel does take place after the eruption of the tooth.

While agreeing with Leon Williams that fully calcified enamel is a dense inorganic substance, I may state there are also in most teeth areas of incomplete consolidation, and there is a frequent penetration of the enamel from the dentine surface by fine prolongations of the dentinal tubules. The evidence is, however, very strong that fully formed enamel is a dense inorganic substance, and that the penetration of the enamel by organic matter is more an accident of imperfect calcification than a characteristic of the well-developed tissue; while, therefore, the fully completed, perfectly formed enamel is virtually inorganic, being fully impregnated with the lime salts, it shows, by the longitudinal lines still visible in some cases in the columns of the prisms, that it was built up on an organic foundation, the protoplasmic strings of the Tomes's processes which have become involved in the calcification. These were first discovered in enamel by Dr. Andrews, of Boston. In most examples of human enamel, however, this process is not complete in every part, but, especially at the dentine junction, shows defects of structure, and often penetration by fine dentinal tubes. This enamel margin having been the first part of the tissue to be laid down might be expected to be the most perfectly calcified, but the fact that it is not is capable of explanation. I have shown that in marsupial enamel a very wide area of organic matrix is laid down while the process of calcification is not at all far advanced. Although the

dentine is, as in all mammalia, the first dental tissue to calcify, by the time that a narrow zone of dentine is formed, a very much wider area of enamel is laid down. This disproportion is not visible in the tooth-germs of any of the higher mammalia I have examined where the area of formed dentine remains wider than the enamel, during the development of the tooth. This part of the enamel matrix, then, is left behind, as it were, by the receding ameloblast cells, and calcification proceeds slowly within it, the interprismatic spaces remaining wide open and enclosing large calcospherites, which can often be seen arranged in a regular row at this part of the enamel. Although the calcification in human teeth is much more complete, in this region it still shows signs of the marsupial condition and imperfect completion of the calcifying process, so that in the spindles, which I look upon as due to enamel defects, and in the penetration in places by dentinal tubes, it shows a tendency to reversal to the marsupial condition. It is also possible that in the early stages of deposit of the enamel the cells have not attained their full secreting function. I think also the evidence is very strong that on eruption the enamel is not fully impregnated with lime salts at its periphery. I have a specimen cut by the Weil process (which I am showing under the microscope) stained with aniline blue-black, in which the enamel, which is of good quality, is stained from the surface some little way into its substance—it is noticeable that the stain is in the substance of the prisms, indicating that not only the interprismatic substance but the prisms themselves are not fully consolidated.

In conclusion, there are two points in the structure of the enamel, as shown in that of the elephant and the wart-hog, to which I should like to draw attention.

I have been unable to find any photograph or drawing of the minute structure of the enamel of the elephant. There is no such drawing or description in Owen's "Odontography" or in any text-book I have examined, but I am sure it must have been recorded somewhere, as the structure is very remarkable and beautiful. The enamel in oblique or nearly transverse section appears to be made up of segments, which overlap one another like the scales of a fish. In longitudinal section the transverse markings which one would expect to be caused by these imbrications are very faintly visible, the prisms appearing to have a wavy or crenate margin. One very peculiar appearance in these sections is a fringing or fimbriation of the margins of these scale-like portions. Many of these show radiating lines, and the radii in some places break up at the margins into feathery processes. The body of the



FIG. 9.

Transverse section of enamel of elephant. ($\times 800$.)



FIG. 10.

Enamel of *Phacochærus*, showing branching of the columns and intermediate columns. ($\times 800$.)

prisms shows a fine granularity. The appearance is so totally unlike that of other mammalian animals that one wonders it has not figured in the text-books. By comparison with the other slides of enamel I am showing, taken under the same magnification, the enamel prisms in the elephant are seen to be much larger. The photograph is from a very beautiful large section given me by the late Professor W. D. Miller, one of the series of large sections which he ground on his electrically driven stones. These preparations are so thin that they can be easily examined with the $\frac{1}{2}$ immersion lens with which these photographs were taken. The section which I will presently exhibit in the epidiascope also shows a very interesting point of structure, attention to which was first drawn in a short paper published in Berlin by Dr. Miller and Professor Dieck. The enamel is held together by long projecting thorns from the cementum on one side and the dentine on the other, forming a stronger union to resist strain than is seen in any other enamel.

The other enamel to which I wish to draw attention, the minute structure of which I do not think has hitherto been described, is that of the wart-hog, also from a preparation by Professor Miller. To this creature, which, justifiably, has the credit of being the ugliest animal in creation, I feel we owe a debt of gratitude, for he appears to have cleared up a problem in the histology of enamel. The compound molar of the wart-hog is built up of denticles which are ovoid, so that there is a much larger area of enamel at the circumference than at the dentine margin, an exaggeration of the condition in human enamel but affording the same problem—the wider area at the circumference—where the enamel prisms appear to be in as close apposition as at the dentine. In this enamel the prisms are seen to branch and supplemental prisms are introduced. Dr. Pickerill endeavoured to account for the fact that human enamel was wider at the circumference than at the dentine margin by a measurement of the diameter of the prisms, and in several examinations found them to be wider at the circumference than at the dentine. He concluded that the individual prisms are conical, with the apex of the cone towards the dentine. Pickerill says he has never seen a prism branching, and there certainly is no record of such an observation in human enamel, although supplemental prisms are said to exist; owing to the complicated twisting and spiral course of the prisms in human teeth it would be very difficult to detect. In the wart-hog the course of the prisms is very direct and simple, and the branchings are distinctly brought into view. Whatever

the bearing of this observation may be on human enamel, it shows that such a method of meeting the difficulty does occur among the mammalia.

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DISCUSSION.

The PRESIDENT: Mr. Mummery's researches into the histology and development of the hard and soft tissues of the teeth have for many years given him an international reputation. His great scientific ability and perseverance have placed him in a position in this field which is second to none in the whole world. He is not only a past master in all the technical processes of research work, but is a brilliant interpreter of the appearances brought out under the microscope; and very careful and cautious in arriving at conclusions, especially when his results are at variance with existing views. His investigations give us a much more intelligible and logical idea of calcification of dentine and enamel than we had before, and go far to prove that it is largely a physical process and is similar in some essential principles to the calcification of lower structures such as the lobster-shell. The calcific material is passed from the blood indirectly to the colloid matrix substance by dialysis

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through a membrane. The calcospherites first formed contain chiefly carbonate of lime and are evidently of no great density or strength, and they break down and disappear on receiving large additions of phosphate of lime, which converts them into the very hard and dense tissues of dentine and enamel as we know them.

Mr. F. J. BENNETT: I have some hesitation in criticizing because I am not a believer in the influence of colloids on crystal formation, and though I fully agree as to the value of Mr. Mummery's recent discoveries in the formation of enamel, I think that the explanation of these appearances is apt to be influenced by preconceptions.

Mr. MUMMERY briefly replied.

Odontological Section.

President—Mr. H. BALDWIN.

(January 24, 1916.)

The Significance of the Radiographs of the Piltdown Teeth.

By W. COURTNEY LYNE, L.D.S.Eng.

(Communicated by MONTAGU F. HOPSON, L.D.S.Eng.)

THE recent publication of Mr. Hopson's extremely interesting lectures to the students of Guy's Hospital on "Apes to Modern Man" and the more recent publication of Professor Keith's work on "The Antiquity of Man" seem to afford an opportunity for further discussion of a very debatable subject—viz., the character of the caninism displayed by "Piltdown man," and I propose criticizing the present accepted view from an aspect which has had rather scant attention hitherto.

It is within the knowledge of all who have followed the discovery of the Piltdown remains that the original find included a portion of the right side of the mandible, with the first two molars in situ. There were certain indications, so it appeared to the gentlemen responsible for bringing the remains of Piltdown man before the learned societies, that a raised canine, of a somewhat anthropoid nature, must belong to this mandible; and although this view met with some criticism at the time, yet, in August, 1913, a raised canine, with a *prima facie* right to a Primate label, was found not very far from the position of the mandible, and Professor Underwood immediately came to the conclusion that "the canine" of this mandible had been found [1]. The British Association, meeting in Birmingham that autumn, provided an opportunity for displaying this canine, and later both Dr. Smith Woodward [2] and

Professor Elliot Smith [3], in various publications, adhered to this belief of Professor Underwood's. More recently, further publications by Professor Gregory and Mr. Hopson accept this view also, on the evidence to hand at the time. Dr. Smith Woodward was rightly cautious, however, before the Geological Society, in that, although he personally believed Professor Underwood to be correct, he yet left loopholes of escape in his references to it, in case of error.

Up to this stage it is possible to have a certain amount of sympathy for the position in which some of these gentlemen are to be found. But now the trouble arises. A book is published dealing with the "Fossil Remains of Man" [4], and in this official guide all doubts are apparently over, for we are told definitely (the words "almost certainly" have now acted in accordance with a well-known law, and become permanently segregated) that the canine found is "the canine" of this mandible, and all illustrations relating to it are based on this presumed fact, and much of the literature is accordingly permeated with this idea. It is because of this positive attitude adopted, apparently without sufficient corroborative scientific evidence, that I beg leave to produce evidence in favour of the incompatibility of this canine with these molar teeth, and it is upon the anatomy of the remains that I choose to submit evidence. Moreover, Professor Elliot Smith has already laid down a law for our guidance, and I cannot do better than quote it. He says [5]: "*It is the business of science relentlessly to scrutinize all the evidence it uses, and not to build up vast speculations from material that will not stand the most elementary tests of stability.*"

It is nearly two years since Dr. Smith Woodward kindly allowed me to examine the relics of Piltdown at South Kensington, and although my evidence must necessarily be in the nature of a severe criticism of his views, I gladly acknowledge his courtesy at all times.

A growing doubt as to the compatibility of these teeth in one individual came upon me, but for a long time I was helpless to achieve any progress with evidence, since Dr. Smith Woodward—in spite of the fact that I demonstrated both to him and Professor Keith the value of the polariscope in this particular instance—could not see his way to allow a section of the canine to be made. It therefore remained unverifiable (and, of course, equally irrefutable), and it was only in the spring of the year (1915) that I began to realize that, without making any section of the tooth, evidence was nevertheless at hand in the X-ray pictures of the molar teeth published nearly two years ago [6] and of the canine published later [7]. Although it is exceedingly

difficult to determine with certainty the "specific" nature of the tooth (and herein I am aware that I differ from the views of even Professor Keith, to whose kindness I must remain a permanent debtor), yet there is sufficient evidence forthcoming to make the statement of the compatibility of these teeth in one mandible appear perverse of scientific evidence. After these preliminary remarks, it is now possible to deal in a more rigidly scientific manner with the evidence before us.

Before proceeding with an examination of the Piltdown canine in detail, it will be necessary first of all to get a grasp of what may quite well be termed the "life-history of the human pulp," and afterwards we shall see what comparative anatomy of the pulp in other creatures has to say in corroboration or otherwise of the action of these same general laws, to which reference will be made. We will assume it a morphological fact, the truth of which no one with any knowledge of the embryology of human teeth will question, that the enamel and dentine are both originally laid down as soft tissues; that at the complete eruption of the crown of the tooth, calcification, as generally understood, is finished in the enamel, partially completed in the dentine already laid down; and that the growing end of the root is practically all soft mesoblastic tissue, from which the final root will be constructed of dentine and cementum. There is no need to produce any evidence of human permanent teeth under 12 years of age.

In fig. 1 will be seen a series of human premolar teeth, prepared by Weil's process and stained in borax-carmin. These sections will display a condition of pulp which must be very carefully noted—viz., the large size of the pulp just after complete formation of root; the relative proportions of dental tissues; the projection of pulp into the crown of teeth; the lack of thickness of cementum; these are points to bear in mind. Proceeding to view the stages as portrayed by these sections in advancing years, it is quite obvious that the human pulp shows a gradual diminution of size with age, a fact perfectly well known clinically by dental surgeons, and borne in mind in all root operations. I also produce three sections of temporary teeth, indicating that a like process also goes on in them. Lest it should be inferred that the canine tooth differs from the premolars, I have produced a series of canines, ranging from 14 years of age to 61, seen in fig. 2, and observe that the young canine possesses a large pulp, and the older teeth pass to almost complete obliteration of the pulp. An antero-posterior view of a canine is shown in fig. 1, and in the same illustration a canine 23 years of age with a fairly large pulp, in order

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that I may not be accused of purposely evading criticism, or suggesting a one-sided aspect in the life-history of pulps. But even in this tooth I would draw attention to its lower third, where conditions prevail both of size of pulp and relative dental tissues which are fairly diagnostic of age; and as a taurodontic condition (to borrow Professor Keith's word) appears associated with a neurotrophic influence on

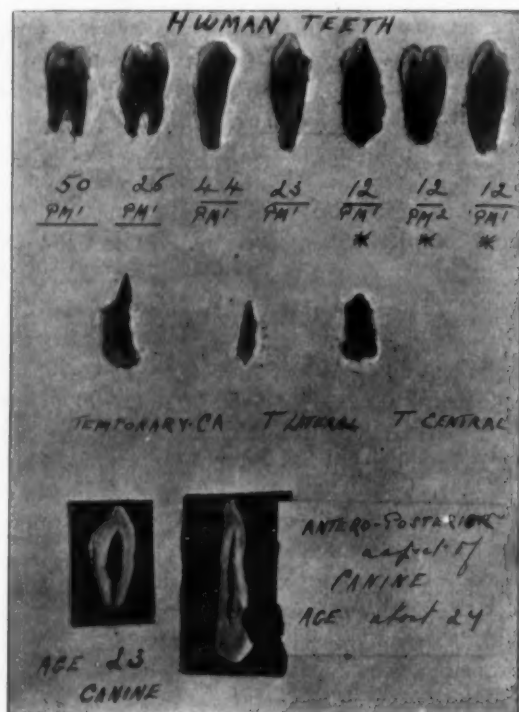


FIG. 1.

odontoblast activity, we occasionally see in some modern teeth with a similar tendency, or with delayed eruption, local dilatations of the pulp. I have observed it in dilacerated teeth. But let it be remembered that there is no evidence of taurodontism in the Piltdown teeth, and the age of my canine (23 years) is even then many years below that accorded to Piltdown, and, I repeat, the lower third is a sufficient index in itself.

In fig. 2 a similar series of the pulps of molar teeth, with equally corroborative evidence of the diminution of the pulp, is shown.

Through the courtesy of Mr. John Humphreys, of Birmingham University, I exhibit a photograph of a section across an inferior maxilla with pulps exposed, and again evidence of a like nature is seen. To what conclusion can we come as a result of the study of these preparations? Surely, there is none other than that "the seven

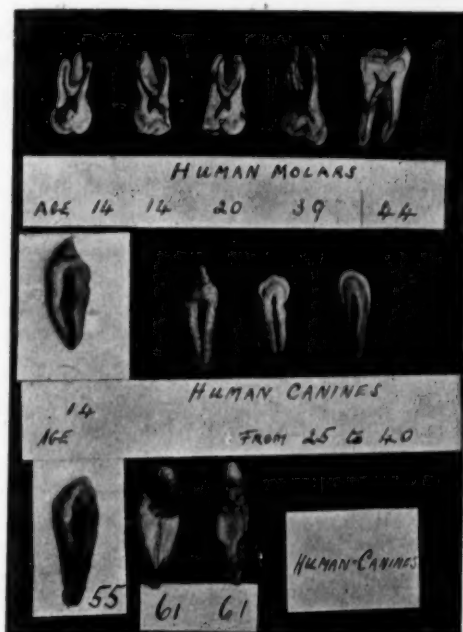


FIG. 2.

ages of man," as portrayed by Shakespeare, may not inaptly be applied to the human pulp, that it is not a fixed organ, but at ages which may approximately be known the human pulp is in certain stages of growth or decline, and that this law in human teeth is as irrefragable as the law of gravitation or conservation of energy is in the world of physics.

Now let us turn to comparative evidence. What has the palæontological record to say? Let us go back to the dim and distant age

of the Old Red Sandstone, and turn to the pages of one of Hugh Miller's less-known works, "Footprints of the Creator"; and figured [8] there is the same process going on in the teeth of *Asterolepis*, one of those remarkable Ganoids. Turn next to the Permian Labyrinthodonts, and still the evidence is clear. When we appeal to Lyell [9] and look at a tooth of *Microlestes*, remarkable corroborative evidence is again forthcoming in Triassic times. Follow the marsupial remains in Jurassic times, the Cretaceous *Iguanodons*, and finally the placental mammals in Eocene times onwards, even to the cave-dwelling animals of the Glacial Epoch, and nowhere, as far as I am aware, is there any contradiction of this law of diminution of the pulp with advancing years, except in teeth growing from persistent pulps, or possibly amongst some few fishes, which is quite immaterial for our present purposes.

I have left extant animals alone so far. I am fortunate enough to have in my possession two dogs' skulls (photograph shown); in the younger one the cranial sutures are quite evident, in the other the cranial sutures are obliterated—observe the large pulp of the young dog, and then observe the stage to which the pulp finally arrives in an older dog. The further photographs of the fox and other animals show a process of diminution of pulp taking place. The cat, by the evidence of the skull, is quite young. Now I am aware that before we can turn our growing data to any logical effect in connexion with Piltdown man, I must convince you further of the validity of this law amongst Primate mammals—other than man, whose evidence we have taken already—since it is here that my evidence must be conclusive. In order to produce this effect I have again to thank Mr. John Humphreys for generously placing at my disposal his excellent museum at Birmingham University for the purpose of securing a series of radiographs. Generally speaking, in the following plates the X-rays were directed on to the lingual aspect of the specimens, in order to obtain the best picture possible of the canines, and as the radiographs of the Piltdown canine published [10] give both aspects of its pulp, a comparison can be made accordingly as the necessity for reference occurs. A photograph is shown of a mandible of *Lemur mongos*; the molars are worn, incisors slightly—but note, even so, the condition of the caniniform premolar pulp. Fig. 3 (a *Semnopithecus*) is excellent evidence regarding the canine pulp once more, and in this specimen the wear on the molars is very slight, and we cannot be dealing with an old individual.

In the mandible of a very young macacus (the Bruh) (photograph shown) the wisdom tooth (M^3) is unerupted (in the upper jaw of this animal the canine—not figured—is not completely formed). I have purposely raised the lower canine above the mandible in order to display the large pulp from an interstitial aspect. There is no appreciable wear upon the teeth of this specimen. Very similar conditions are represented in the photograph of another macacus—viz., M^3 just erupting, no wear to speak of, and the canine showing a large pulp chamber just beginning to close.

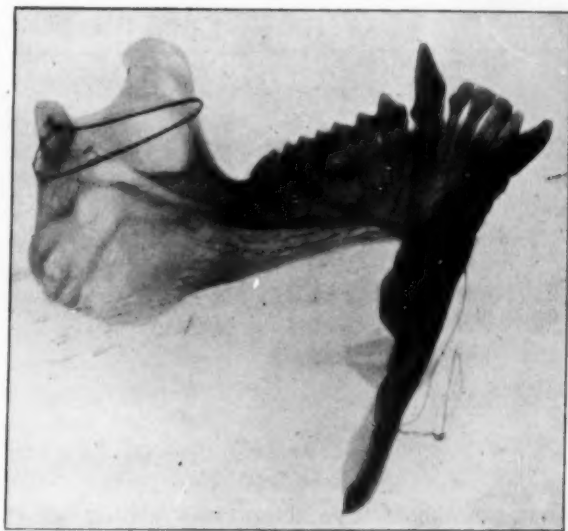


FIG. 3.

Fig. 4 shows above *Cynocephalus babouin*, with a full dentition, and very slight wear on the teeth; but observe how the canine pulp is displayed by a thin white line. Below is the mandible of a female baboon; observe that the teeth are well worn, and that the canine pulp, such as is shown, is evidently still further diminished.

In the pulps of a very young *Cercopithecus* (photograph shown) the third molar is not erupted, and such of the pulp as is displayed shows an increasing size in its alveolar portion, as we should anticipate in a tooth just nearing complete eruption.

In the view of the superior maxilla of a young female gorilla, with molars very little worn and cranial sutures perfectly patent, is observed the comparatively large pulp in this young example, as we again anticipate. Obviously, too, the wisdom tooth (M^3) has not been erupted very long.

In fig. 5 is represented the mandible (one half) of a chimpanzee, with molars rather more worn than those of Piltdown, and cranial sutures indistinct. These are anthropoid canines that cannot possibly have had anything like the years of wear of the assumed *Eoanthropus* canine, and yet the condition of the pulp in its comparatively diminutive size is self-evident.

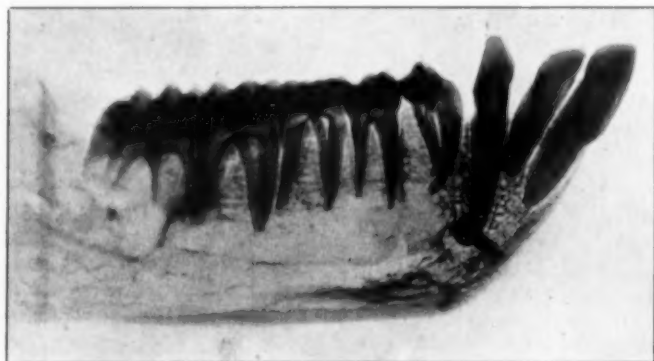


FIG. 4.

In what is, probably, a male chimpanzee (photograph shown) there is wear rather exceeding the Piltdown molars, and although there is a moderate pulp in the canine, yet it is not comparable with the condition we shall see later in the Piltdown canine. The portion of pulp in the crown, the relative proportion of tissues, the apex of the root closed, by probably a considerable portion of cementum, are points to notice.

I have purposely sought for this evidence concerning the chimpanzee's teeth, since the writers of some publications regarding *Eoanthropus* never weary of insisting on the likeness of the Piltdown mandible to that of a chimpanzee jaw; unfortunately for them, dental comparisons simply prove that the chimpanzee and the orang-utan also obey pulp laws similar to those obtaining in man, and Professor Underwood's own X-ray of a chimpanzee mandible [11] is excellent.

corroborative evidence from the point of view I am insisting upon, but unfortunately for Dr. Smith Woodward it does not display the apical conditions to which we shall refer again when dealing more directly with the Piltdown canine.

The evidence from all these Primates, then, as also that of Heidelberg man [12], as indicated by his pulps, and that of Krapina man [12], as shown in his pulps, is clearly in harmony with all the previous evidence, and declares that any living organ, such as the pulp, is governed by certain laws of life. Finally, however, let it be observed that the molar teeth of Piltdown man (*Eoanthropus*) indicate, by the published radiograph [13], the universal working of this law of life.

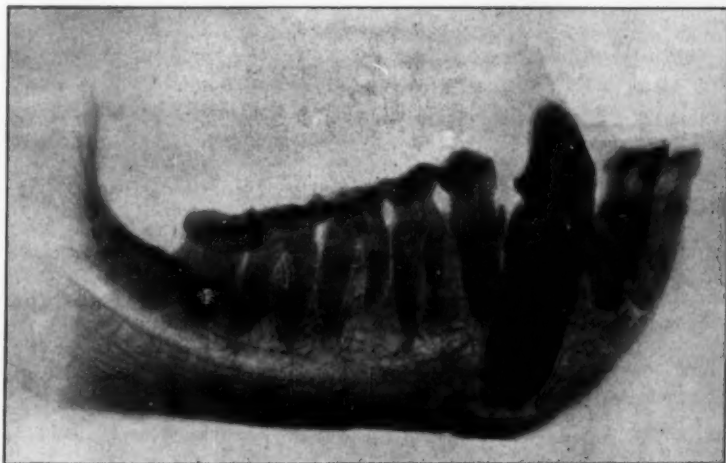


FIG. 5.

Consequently, it is now appropriate for us to assume it as proven that certain conditions of the pulp appear at an approximately fixed time in the various creatures, and, as a necessary corollary, that all the teeth of limited growth of any one creature must be subject to the same law. And if the evidence of separate fossil or recent teeth places the ages of the teeth by their own evidence far apart, they could not be the property of one and the same individual, since no creature can be two different ages at one point of time. Having, then, arrived at this axiomatic condition, we will refer to fig. 6, a tracing of the general outline

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of the pulp cavity in the Piltdown canine; for excellent radiographs I must ask the reader to refer to the source already indicated. Before proceeding, however, let me give some views regarding this tooth, and the molars of Piltdown also, as expressed by the following investigators: Dr. Smith Woodward [14] described the two molars as "essentially human and worn flat in human fashion by the mastication of coarse and gritty food, &c."; Professor Underwood [15], speaking of these same teeth, says: "*They are worn down by use to such an extent that it was impossible that the individual could have been less than 30 years of age,*



Tracings of Piltdown canine (pulp). After radiographs in the *Quarterly Journal of the Geological Society*, April, 1914.



Tracings of Piltdown molars (pulp). After radiograph in the *British Journal of Dental Science*, October, 1913.

FIG. 6.

probably a good deal more"; and Professor Elliot Smith [16] remarks concerning the cranium—and, remember, he accepts the finds as those of one individual: "*The suggestion is that the sagittal suture had recently closed when Piltdown met his death; this may occur in modern man at any age between 30 and 40 years of age, although in rare instances it may even occur before 30, or (more often) be delayed beyond 40.*" More-

over, as Dr. Smith Woodward pays tribute to Professor Underwood's aid, and has never, as far as I am aware, dissented from these views of Professor Elliot Smith and Professor Underwood, I think, therefore, that with these previous quotations I am representing fairly these gentlemen in stating that they accept the age of Piltdown man as 30 years or over. Certainly the evidence (apart from the canine) indicates apparently an age about—or, in my opinion, a shade below—30 years of age. Therefore, in looking at any teeth erupting at about 11 years of age, as suggested by Dr. Smith Woodward [17], and still the property of Piltdown man at death, we must see a canine having had twenty years or so of supra-alveolar life, and having been exposed to use for all this time. Now, I give some views regarding this canine: Professor Underwood [18] says: "*It is quite simian in form and is raised and pointed,*" &c. Dr. Smith Woodward [19] does not *in toto* accept this, for he says: "*Its crown differs a little in shape from any canine of any known ape, and agrees more closely with the milk canine of modern man.*" However, this following astounding statement, viewed in the light of my further remarks, is made by Dr. Smith Woodward [20]. He says: "*The root of the tooth is complete, somewhat deeper than the crown*"; also [21]: "*The extent of this pulp cavity, which is widely open at the lower end of the root,*" &c. One further quotation from the same source [22]: "*No trace of the socket for the tooth is seen at the bone preserved at the symphysial end of the fragmentary mandible.*" Mr. Hopson, I observe, admits the possibility of a minute portion of the apex of the tooth having been broken off, and Professor Underwood has written later [23]: "*It is possible, but by no means certain, that a tiny portion of the apex is missing.*"

It is now appropriate to let the tracings of the Piltdown canine be carefully examined, in the light of all the evidence in the previous plates. Where is the parallel to these conditions shown in any raised canine of any creature 30 years of age or over? Will turning to the temporary human teeth for assistance avail? Will it aid us to say that Eoanthropus was neither essential man nor yet true Anthropopithecus, and therefore not like either one or the other, but rather intermediate? Is this one creature to be a law unto himself? Have we found some ancient "Canute" who forbade the "waves of time" to surge around his canine? Must we not rather be guided in our judgment of the age of this tooth by the nearest parallel conditions that can be found in the nearest creature allied to it? And (assuming for the moment that we are really dealing with a permanent canine of Piltdown man) the

conclusion, then, must be that we cannot see any interpretation which warrants an age for this tooth exceeding 17 years of age—certainly it will be impossible, in spite of ceaseless and subtle reasoning, to make a pulp cavity of this size and nature congruous with 30 years of age!

I cannot see any escape by the theory of "intermediate" conditions between Simian and true Homo, since some of the investigators having already placed the age as about 30 years, the relatively diminished longevity, say, of an Eoanthropic creature would hasten on calcification, and so quite counterbalance any possible increased size of pulp owing to simian tendencies. Moreover, one has to face the following fact (and here, *mirabile dictu*, Professor Underwood must surely have been labouring under some curious misapprehension, since I am unaware that he has ever corrected Dr. Smith Woodward's implication other than by the uncertain statement of his in the *Dental Cosmos* to which I have referred). This tooth is spoken of as having a completed root widely open at the lower end. We shall await with considerable interest the production of such a phenomenon coincident with 30 years of age. It is a mirage in the minds of ardent theorists or an elusive "will-o'-the-wisp," and nothing more. Nature is exceedingly careful of undue pressure on the apical end of the pulp, and either by continuous formation and calcification of dentine or a plug of cementum, a hard base is not long in being produced, and even then the termination of the pulp is generally at a small foramen not at the extreme point, and in all animals where great strain may come before calcification is well advanced, the canine is curved remarkably, at times in such a manner as to remind one of teeth growing from persistent pulps. Now, however, we are asked to accept about twenty years of a widely open apical pulp-end resisting the wear and tear of probably hard food.

Again, will any anthropoid or human canine show the same proportion of formed, and to be formed, dentine in the lower half, say, of the root at 30 years of age? Examine measurements transverse to the long axis of this tooth, and see whether a nearer parallel in tissue proportions is not to be found in the premolars marked by a * in fig. 1. We know, moreover, that at the stage of life when dentine formation is heavy and continuous, that the pulp is a perfect network of ramifying capillaries supplying the odontoblasts, and how are we to account in comparatively full adult life, or even older still, for finding very heavy dentine formation yet to be accomplished at a time when the general decline of the vascular system is setting in? Further, the

risk of pathological degenerations in a pulp this size at this age makes such a condition *ipso facto* unthinkable. The gradual closing of the pulp cavity with advancing years becomes a physiological necessity.

It is vain to transcend knowledge, put aside evidence, and play with one's mental faculties, and before the congruity of the Piltdown teeth is a proved biological fact, these questions will have to be satisfactorily answered by evidence from the laboratory of life, and unless this is forthcoming there is nothing for it but to withdraw from an apparently untenable position. But as I have no desire to shun a method of criticism so well established by Charles Darwin, we will examine some possible objections directed against my attitude of probable incongruity of these teeth.

(a) It may be suggested that the root of the canine is, after all, fractured and a portion missing. But if this canine be lengthened at all, according to published drawings the impression for the same should be seen in the alveolus, and Dr. Smith Woodward already has told us that it is not there. Moreover, even if one-third of the root were missing there would still be remaining incongruity of tissue formation for this age; neither will one suggest raising this canine one-third higher, for when we come to the upper canines of a male Piltdown creature, this condition with the glenoid fossa would be rather curious. To urge such an objection as this is to steer the craft away from Scylla only to wreck it on Charybdis.

(b) Occasionally we see, say in two teeth, a larger pulp in the older tooth, but when this is so there is either a taurodontic condition present or the age of the teeth is fairly nearly approximate, and however careful one might have to be regarding this in some cases, it does not affect the argument regarding Piltdown man, since the molar radiograph indicates normal conditions, and the age of difference between these teeth and the canine is altogether too great to be bridged in this manner.

(c) Mr. Charles Dawson's objection [24], "*that there is nothing in their mode of occurrence suggesting different individuals,*" is worthy of note. Surely no one conversant with palæontology will argue that all the teeth found within a 15-ft. radius of deposition need of necessity belong to one individual? It would weary the reader to take him through the numerous records, time after time, of isolated teeth found, with sometimes as much as twenty years elapsing before a more complete discovery has been made. The reasons for this are not

obscure when one remembers their hardness and the ease with which they will drop out from the alveoli at times. Besides, is it not more reasonable to expect M^3 to turn up, the tooth belonging to the part found, than the wanted tooth of the missing place, the arithmetical chances of finding the same being one to twenty-nine against all the rest of the teeth of this individual. This very "Guide-book of South Kensington" [25] speaks of one tooth of *Macacus pliocenens* found in brick-earth at Grays, in Essex; and to this day the Taubach remains are only two or three teeth, and *Pithecanthropus erectus* is a somewhat similar case, the certain unity of these teeth in the one individual being called in question by this same Guide-book [26]. It is in connexion with these isolated finds that I can see a most important future for the X-rays in the hands of those skilled in the knowledge of the life-history of the teeth of various creatures. Parenthetically I would remark that Sir A. Geikie [27], along with others [28], has some idea as to *Microlestes* being a Monotreme. If I read aright Lyell's [29] illustrations, we are dealing with a very recently formed second dentition tooth, and as we have no evidence in present Monotremes of any second calcified series, I am much inclined to look upon *Microlestes* as marsupial in type, and we may have to place the Monotremes as a degenerate divergent subclass.

(d) To return to Piltdown. "Wear and tear" is another stumbling-block brought forward. Anyone fully conversant with animal mouths knows that harmony of wear in their teeth may or may not be present; quite recently erupted teeth, too, will often show a fair amount of erosion. But my final remarks will deal more forcibly with this objection.

(e) The *prima facie* reasons of the mylohyoid groove and the effect of the superposition of Piltdown's jaw on Heidelberg's [30], showing excessive length forwards of Piltdown's, is another objection. But one must remember that *prima facie* reasons are not final proof; a distinction must be made between certainties and plausibilities. I might argue that on *prima facie* grounds the contour of the glenoid cavity contra-indicated a raised canine, and that, the act of yawning with such articular mobility and with projecting canines would be one which Piltdown man would not be likely to indulge in with pleasure. Again, as regards the superposition of the jaws, surely the possibility of fallacious reasoning must be evident to any close observer. If we fractured an "underhung" jaw of one person and superimposed it upon a similarly fractured "agnathic" jaw of another person, placing M^1 over

M₁, and reasoned according to this code, we should be asking for chances of error.

(f) The condition of large pulps in modern temporary teeth (with its implication of a certain palæontological law) is another citation of critics who have not observed that this is, even in these teeth, but a passing phase, and that after a while the terminal apex of root does undergo the same changes as in permanent teeth; and as the pulps are generally seen within a few years after eruption they are necessarily rather large proportionately.

(g) Another critic will say: "But you imply the coexistence of a humanoid anthropoid at Piltdown." I suppose that there is no known reason why the remains of a recent human being and traces of a modern anthropoid might not be found together? If the canine found be not the canine of this mandible, it is apparently either that of another Eoanthropus or a humanoid anthropoid, and I shall deal with its Eoanthropic character later.

(h) Another person suggests, "May not pathological and solvent conditions have produced this state of pulp cavity in Piltdown?" I have seen an enlargement near the apical end of the pulp canal in necrotic roots, but never in such an even contour, or extending other than a short distance.

Finally, then, to sum up, we arrive at this conclusion: Absolute anatomical evidence (and this is the final court of appeal) appears to invalidate the congruity of these teeth in the same mandible, and *prima facie* objections cannot remove this indubitable evidence, even if my replies to the objections fail to convince some who may still have "an affection for doubts." If this tooth be incongruous in this mandible, no power on earth can alter the fact that "the canine" has yet to be found, and is at present hypothetical.

But, however, a canine of a curious form has been found, and can we get any further light on its "specific" nature? There are points concerning it that appear to me to have escaped notice, and their significance has thus been overlooked: the very thin layer of enamel displayed, the loss of the tip of the tooth, the outwardly bent tendency of the root with apparent absorption beginning at the basal end of the root, along with the large size of the pulp, all point to a deciduous canine, and almost certainly a Primate one. The "wear" on the tooth is now a favourable argument, since the extreme thinness of enamel and less highly calcified tissues would be conducive to rapid abrasion or erosion. I have seen this type of "wear" in embryo, so to speak, in

very many temporary teeth; it is produced in the process of extruding the temporary teeth. It also becomes quite rational on this hypothesis to understand why a single tooth should be found, and the temporary canine is often retained beyond a short period even in the mouths of young anthropoids; the temporary canine of an orang may be found in situ when the second permanent molar has come into full use. The general outline of the Piltdown canine is not harmonious with the view of a permanent tooth. These teeth have a bow-shaped curve towards the tongue, and especially is this so with raised canines; but in this particular tooth if a line be dropped from its cervical margin or a little beneath on the labial aspect, when in an upright position this line will be seen to almost bisect the outer base of this tooth—an occurrence quite common amongst deciduous teeth if similarly viewed, and for which there is a reason. Again, I repeat, the amount of wear, the shape, and the pulp cavity harmonize in a deciduous tooth, but are necessarily contra-indicatory in a permanent one. At this point it is convenient to refer to a radiograph representing the deciduous dentition, only just completed, of a very young chimpanzee. Observe the parallel to the Piltdown canine; but this chimpanzee is seen at a very early age. In turning to fig. 7, however, we see a later stage in the deciduous dentition of a young orang, with the first permanent molar well in situ, and here is shown an extraordinary likeness to the pulp of the Piltdown canine. It is in the stages of the life-history of a deciduous Primate tooth that we must seek for a near parallel, and had Dr. Smith Woodward taken an X-ray of the orang's temporary canines instead of comparing their external form only [31], it is inconceivable that he could ever have been quite so dogmatic.

Both these X-ray pictures show the forming permanent teeth, and one can easily see how absorption of the apical ends of the temporary teeth, coincident with the eruption of permanent ones, would leave the former with a pulp widely open at the lower end. Doubtless the size of the Piltdown canine will be urged against its being a deciduous tooth; but again this is mere *prima facie* argument against direct anatomical support. And one must not forget that amongst the Lemuridæ in late Pleistocene times, huge forms arose (viz., *Megaladapis insignis*), and it is quite conceivable that larger anthropoids than we have living now were in existence in late Pliocene or early Pleistocene times, and a somewhat large deciduous canine would thus not be irrational. Probably our extant anthropoids are more distant cousins than some earlier forms, which are at present unknown to us. Granted this

deciduous character of the Piltdown canine, the question arises, To what Primate creature must we assign it? If as such we seek to harmonize it with *Eoanthropus*, I fear that we have difficulties to overcome. Presuming it to be a deciduous tooth of an *Eoanthropus*, are we likely to find the permanent canine any smaller or any shorter, and how are we to get such a tooth in situ in a mandible such as Piltdown man possessed without evidence of the socket for the same? We know that the evidence of anthropoids with raised deciduous canines points to much more projecting permanent ones, and I hardly think the

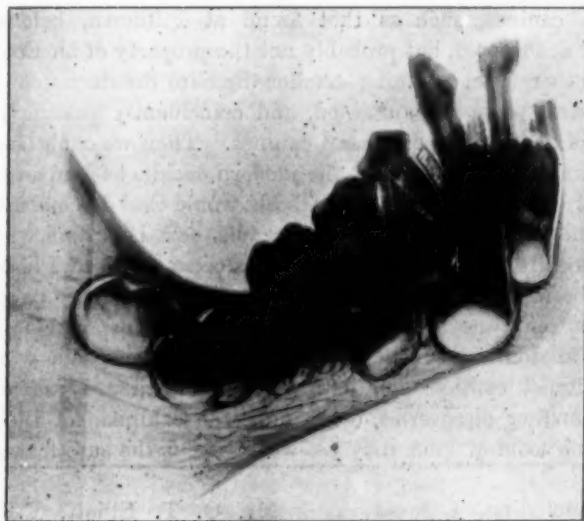


FIG. 7.

reconstructors of Piltdown man are prepared to model this individual with raised canines perhaps exceeding those of any known anthropoid. Moreover, sufficient emphasis has not been laid on the very thin layer of enamel displayed in the Piltdown radiograph. To claim this tooth (even though it be a deciduous one) for an *Eoanthropus* is to claim it for "true man," and neither the deciduous nor permanent canines of modern man in any way corroborate this proportion of enamel and dentine; in evidence of this, I show a photomicrograph, taken with polarized light by crossed prisms, of a human temporary canine, in

which the enamel is certainly not insignificant on the labial aspect, and the proportion of enamel to dentine in the permanent canine is even greater still. A reference, however, to fig. 7 shows once more how near an approach in enamel proportions there is between this temporary anthropoid tooth and that of the Piltdown radiograph. If, then, we accept this canine as deciduous and cannot correlate it satisfactorily with Piltdown man, all that we can do at this stage is to theorize, and to me the following way out of the maze of doubts is to postulate, in late Pliocene times, a large anthropoid showing humanoid tendencies, and with moderately raised canines (? *Pithecanthropus erectus*); and branching off from this we have one type still maintaining fairly large deciduous canines, such as that found at Piltdown, belonging to a humanoid anthropoid, but probably not the property of an Eoanthropus, and the other type, retaining smaller (human) deciduous canines, with the ancestral pattern maintained, and coincidentally passing on to the eruption of non-raised permanent canines. Then we could see how the canine found at Piltdown and the modern deciduous human canine are somewhat alike in shape, since both would be descendants of one common near related ancestor. He would be a bold man who asserted that all anthropoid types in early Pleistocene or late Pliocene times had yet been revealed. However, this latter part of my paper I admit frankly, is only one more hypothesis as to man's possible phylogeny. But the anatomical evidence regarding the other facts will, I trust, at least impel cautious anatomists to assume a *sub judice* attitude, pending further discoveries, concerning the caninism of the Piltdown man. The loom of Time may yet weave for us the sure features of this creature.

In concluding, I must thank Mr. O. T. Elliott, F.R.M.S., of Birmingham, for his kind assistance in arranging for the radiographs (by Messrs. Watson and Glover) and for his own photographic work.

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DISCUSSION.

Dr. SMITH WOODWARD: It is certainly very gratifying to Mr. Charles Dawson and those who have shared in interpreting this discovery to feel that it has aroused such wide interest and stimulated so many researches connected with the subject. It is important that the questions involved should be looked at from every possible point of view because, as in nearly all cases in which we depend upon fossils, we have, at any rate at first, to balance probabilities. When we make discoveries of the nature of Piltdown man, we have only imperfect evidence to deal with, and we cannot do much more than arrive at probabilities. The more the points of view from which we consider the subject therefore, the more satisfactory the result. One of my friends in America has lately said that the lower jaw under discussion does not belong to the skull, but really represents a new species of chimpanzee. We are told to-night that the canine tooth cannot belong to the same animal as this jaw; and so we now reach the conclusion that the Piltdown fossils represent three distinct animals. It seems to me most improbable—almost incredible—that when we find a unique Primate skull in the same place as an absolutely new Primate jaw, and close to an entirely new Primate tooth, we are dealing with the remains of three distinct animals; especially when we remember that we are accustomed, among fossils, to find combinations that are strange to those who study only the modern world of life. I still think that all the researches which have been made really leave us a very wide range of possibilities from which we can select; and as, by referring all the Piltdown remains to one animal, we are simply producing another illustration of a law which has been repeatedly proved to apply to various races of extinct mammals, I feel we may have confidence in our inference. We must also remember that, if we put these remains together, we realize the prediction which was made, on other grounds, by Professor Elliot Smith at the Dundee meeting of the British Association just before the Piltdown discovery. I therefore think there is not such an inherent improbability that all the Piltdown remains belong to one individual as might appear from researches such as those which have been detailed to-night. I wish especially to add that all the specimens under discussion have now been given by Mr. Charles Dawson to the British Museum, where they are completely accessible for study; and I hope all who are interested will examine them and assist in arriving at the truth.

Professor ARTHUR KEITH, F.R.S.: It gives me great pleasure, Sir, to be here to-night and to be able to congratulate Mr. Courtney Lyne on a very able presentment of a very difficult problem. We have to distinguish very sharply between the facts which he has added to our knowledge and the new explanations which he has offered us for some of those facts. The new facts which he has brought before us are his studies in the formation of dentine—

namely, that the dentine is being formed all through life, and that therefore the pulp cavity, taking the average of one age with another, is steadily decreasing. I do not know how far dentists had recognized that fact before; personally, I had accepted that as a general truth, but I did want observations on a very wide series of teeth at different ages to illustrate that truth. Mr. Lyne has to-night brought a very large series to support that contention—namely, that the pulp cavity decreases in size during life, and, *a priori*, a large pulp cavity indicates a tooth from a young animal and a tooth with a small pulp cavity is probably from an old animal. I must not detain you too long, but the identification of the original owner of the Piltdown canine is, to me, an extremely interesting problem, and has been since the first discovery of the Piltdown remains. Perhaps some of you will recall that when Dr. Smith Woodward first announced that this animal from Piltdown certainly had a large projecting canine I questioned it, and said I was almost certain it could not have had a large canine of the Simian form. The grounds on which Dr. Woodward based his inference were that the Piltdown mandible possessed the outstanding features of an ape's jaw, and that these features—the kind of symphysis, the mylohyoid ridge—always had been correlated with a projecting anthropoid-like canine tooth. That, I think, was the basis on which he made his inference; he was certain that when the Piltdown canine tooth was found it would be found to be shaped like an ape's. I based my conclusion on the form of the glenoid cavity; the Piltdown kind of glenoid cavity I knew of only in the human skull, and regarded its form as associated with the movements of the human jaw, to which our glenoid cavity is adapted. I could not, therefore, conceive of the presence of a projecting canine with the human form of glenoid cavity, because with such a tooth the jaw could not be moved easily from side to side in ordinary mastication. It was on those grounds I inferred that when the canine tooth was found it would be found to be a big tooth, but not a projecting canine. A tooth was found, and I had an opportunity, at an early period of the inquiry, of seeing it. It was not, as it were, to my interest to accept a projecting, ape-like, canine tooth; it was rather to my interest to refuse to accept the canine which Dr. Smith Woodward had discovered. When I looked at it I saw that there could be no doubt of it belonging to an extinct member of the higher Primates; it was less humanoid than anthropoid; all its characters pointed to it belonging to a creature half-way between man and ape. There was one criticism which I made at the time—namely, that the amount of wear exhibited by that tooth seemed to me out of keeping altogether with the amount of wear on the molars. I formed the opinion from the condition of the molar teeth that the animal was just mature. But that is not a particularly safe criterion to rely upon as to age. I expected a canine tooth which was slightly worn. It was not only the degree of wear which made me hesitate to accept the tooth as of that of the Piltdown jaw, but it was also the kind of wear. I do not think Mr. Lyne has referred to that aspect much to-night; but the way in which the canine tooth of the Piltdown creature is worn is peculiar. In all the

anthropoid teeth I know there are two facets. As the jaws work, the lateral incisor makes the biggest impression on the corresponding lower canine. But the corresponding upper canine tooth also wears a facet on the lower. I found, on going over our College Museum specimens, especially dentitions of female gorillas, that occasionally lower canines are seen with only the facet, which is worn by the lateral incisor, corresponding to the kind of wear which the Piltdown tooth shows. These two features of the Piltdown canine—the degree of wear and the kind of wear—seemed out of keeping with the other Piltdown fragments, so I had to resolve whether my first law was right, that you could not possibly have a side-to-side movement, projecting canines, and a human form of glenoid cavity in the same animal form. When I considered the type of wear upon the Piltdown canine—which I presume is a lower right canine tooth, although there is now a school in America which places it as an upper canine tooth on grounds which I do not understand—I was forced, as it were, to the conclusion that, with such a type of wear, side-to-side movements would be possible and that Dr. Smith Woodward was probably right in assigning it to the human being he had named *Eoanthropus*. I may say I would not readily forsake a position I had taken up if I found it represented the nearest possible approach to the truth. One wishes to state our inferences so that they will still hold true when we are all dead and gone. I concluded, with all the evidence in front of me, that the probability was that Dr. Smith Woodward was absolutely right; that he had found the missing lower canine of the right side, and that my law was evidently wrong—that is to say, the law which I postulated concerning the form of the glenoid cavity. I had supposed that our glenoid cavity had assumed its present shape as the projecting canine disappeared and assumed a position flush with the surface of the other teeth. Our knowledge of the movements of the jaw is not by any means final. My present difficulty relates to the manner in which the upper canine tooth of *Eoanthropus* articulated with the teeth of the lower jaw. After studying the matter as closely as I can, I have not yet obtained, nor have I yet seen, a satisfactory solution of this difficulty. I simply take the broad truth that, as Dr. Smith Woodward said, these parts were found together, that they are all strange but clearly closely related from a structural point of view. They were part, not of a low kind of beast but of a true kind of man. These parts having been found, one has, as Dr. Woodward says, to exercise one's best judgment.

Before the discovery of the Piltdown tooth, I had arrived at a conclusion which I thought to be of some importance, namely, that in ape jaws the canine tooth is a part of the side series of teeth; in the human dentition the canine has swung round, so that it comes to form not a part of the lateral series, but part of the anterior or incisor dentition. I had inferred that the disappearance of a projecting crown from the human canines had occurred as they were moved from a lateral to an anterior position. I conceive that the wear on this Piltdown canine indicates an intermediate stage in its migration from a lateral to an anterior position. In this manner I seek to explain how it is

possible to associate in the same form a projecting canine and yet a truly human glenoid cavity.

Coming now to a much broader matter, Mr. Lyne to-night spoke of laws. There are, of course, laws *and* laws. The kind of law which he was speaking of to-night I am afraid does not exist. You know the story of Cuvier: that a certain bone was placed before him and that he reconstructed the animal, more or less correctly. That is possible; with a certain part you may be able roughly to outline the animal. But anyone who has worked at the anatomy of animals nearly related to man knows that there are structures which appear to be totally at variance with the general organization of the animal. Take, for example, the liver of the gorilla; we expect the gorilla to have the same form of liver as the rest of the anthropoids. It has not; its liver is lobulated in the same fashion as in the lower apes. If we were to find not a canine tooth but a liver of an extinct and unknown ape, no one would suspect that such a low type of liver could be assigned to such a high type of anthropoid. The gorilla's liver is a flagrant breach of Mr. Lyne's law. I could give you many other instances of the same kind from the human and anthropoid bodies. I think I could select instances from every Primate I know. I could cite a part or an organ, and show you it was incompatible with the general law of organization in that kind of animal. When we are shown, as we have been to-night, a large pulp cavity in a canine tooth, I do not think that is sufficient to make that tooth an impossible member of the jaw in question. The pulp cavities of the Piltdown molars, Mr. Lyne says, are not large. They are not large when you compare them with these of the Heidelberg man, nor large when compared with the teeth of Neanderthal man; but compared with most modern molar teeth they are large. [Mr. LYNE: Proportionately to the supposed age of the creature.] Of course we must allow for age; but suppose *Eoanthropus* is 25 or even 20 years of age. [Mr. LYNE: You must go by other guides than the mere size of the pulp cavity—formed tissue as well; and if you look at a skiagram of the Piltdown molar you will find, particularly in the root region, there is a considerable proportion of formed tissue.] But the pulp cavities in the molar teeth are fairly large, and the pulp cavity of the canine tooth is also large. I do not think it is a character which can absolutely eliminate that tooth from belonging to that jaw; and I take practically the same view as Dr. Smith Woodward, who approaches it from a different standpoint. I am of opinion, at the present time, that the mass of evidence is in favour of the three portions found going together as parts of the same individual.

Professor ARTHUR UNDERWOOD: I have listened to the very interesting paper, and, if I may say so, the still more interesting commentary, and I hardly think, after all, that my contribution is needed. But as my name has been rather freely mentioned in the paper, I would like very much to say a few words. Professor Keith has already, in words better than I could use, said what was really in my mind as I listened to the paper, and I thank your

Honorary Secretary for his courtesy in allowing me to look at the proof of the paper. We all know, and have all taught for many years, that the gradual change of the tooth pulp into calcified tissue proceeds to obliterate the pulp and to reduce it in size. I am, unfortunately, old enough to have taught it for forty years. And I think those who can say as much would agree that he would be a very bold man indeed who would attempt for a moment to say that there was such a rigid law appertaining to such a transformation that he would be able to name not only the exact age of the tooth of a modern man according to the degree of invasion of the pulp by calcified tissue, but also to say exactly what was the age of the individual who lived under conditions certainly very much previous to such conditions as obtain now, when the rules which govern changes may have been different. I argued from the degree of wear and tear in regard to the age, and when I said 30 years I meant it might be a little more. I still think the wear and tear question is very important, and I think the reader of the paper passed rather lightly over the nature of the wear and tear in the canine which we have been discussing. It is not at all the wear and tear which occur in temporary teeth, it is a huge removal of tissue by the wear of the upper canine. It has gone so far as partially to invade the pulp cavity itself. By looking with the microscope at the surface of the tooth, you can see the portion of pulp where the secondary dentine has been formed to resist that invasion. I do not think one can build too much upon the calcification of the pulp; I think it is asking a well-known law to bear too severe a superstructure. The wear and tear question, I think, entirely proves that the age of that tooth is a great deal more than the age which has been assigned to it. I am a little bit in a difficulty about what age exactly Mr. Lyne assigns to it; I think he said 30 years for the molars and 17 was his approximate guess for the canine. [Mr. LYNE: Allowing that the end of the root of the tooth is, as Dr. Smith Woodward has estimated it to be, widely open.] I think Dr. Smith Woodward would not press that point—I do not know. But my feeling is that there is a considerable lump off the end of that tooth, and when I said a "tiny bit" I meant in comparison with the size of the whole tooth. It is very difficult to regard it as a milk tooth, which I gather is Mr. Lyne's final conclusion.

Professor ELLIOT SMITH (Manchester): I came to the meeting to-night rather to hear what others said than to take part in the discussion myself; but I should like to express my thanks to the Secretary of the Section for giving me the opportunity of attending, and for allowing me to see the proof of Mr. Courtney Lyne's paper. I do not profess to have any expert knowledge of teeth; and my views concerning the Piltdown teeth have been based upon general anatomical considerations, arising out of the study of the skull as a whole. On the last occasion when I was present at a public discussion of the Piltdown fragments in London, I also took some small part in combating the view that the canine tooth did not belong to the same individual, part of whose jaw and cranium was found on the same spot; but on that occasion the

contention put forward by Professor Keith was that the canine, being so much worn, was *too old* to belong to the same individual as the molars. Mr. Courtney Lyne also claims that the indications of the canine's age prevent it from being associated with the molars, but on the ground that it is *too young*. The whole of Mr. Lyne's case depends upon this question of age. He stated that I had attributed an age of about 30 years to the Piltdown individual; but he also quoted my exact words, which give no grounds at all for such a statement. I was scrupulously careful to explain that the cranial sutures of the Piltdown skull were in the stage that "may occur in modern man at any age between 30 and 40 years," meaning to imply that there was no evidence to justify one in assuming that the dates of closure of the sutures in modern man could be used for the determination of the age of the Piltdown man. I was particularly careful to guard against such an inference, for during the last fifteen years I have repeatedly called attention to the consideration that, even when estimating the age of such relatively modern remains as those of the ancient Egyptians, it could not be assumed that the facts derived from the study of modern Europeans could be applied to them. Among the large series of Egyptian skulls that I have examined I have so repeatedly found the widest discrepancies between the indications of age afforded by different parts of one and the same individual that the necessity for caution in estimating ages has been very deeply impressed upon me. Under such circumstances I do not attach so much importance as Mr. Lyne does to such discrepancies as he claims to find between the ages of the canine and the rest of the skull. I do not suppose that even in the case of a modern canine he would claim to be able, in any individual case, to estimate the age as certainly being not more than 17 or 18 years. In view of the exceedingly primitive and ape-like features of the Piltdown skull, it is in the highest degree probable that its sutures closed at an earlier age than in modern man. Thus it seems to me that, even if we accept his evidence at its face value, there might not be much more discrepancy between our estimates than four or five years; and if due allowance is made for individual variations even this may disappear. But in dealing with such problems as this one cannot be reminded too often of the fact that we are considering a type of creature which hitherto was entirely unknown. It is rash, therefore, to argue from speculations as to what ought to be found in claiming discrepancies in the features which actually are found. When one considers the fact that many writers are now claiming that the mandible did not belong to the same individual—or even the same genus or family—as the skull, and that Mr. Lyne suggests that the canine was not a part of either, we have to consider the possibility that three hitherto unknown man-like apes, or ape-like men, died side by side; but while one of them left a fragment of his cranium without jaw or teeth, another part of the mandible without any of the cranium, and a third his canine without any fragment of jaw or skull, I claim that the balance of probability—and, as Dr. Smith Woodward has said, it is a question of probabilities that we have to deal with—against this hypothesis of three different unknown creatures

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leaving complementary fragments is simply colossal. But when in addition it is claimed that the jaw is that of an ape, and the canine that of the same or another ape, there is added to this improbability the further difficulty that, so far as palaeontological history is known to us, all the anthropoid apes had been cleared out of Europe by the middle of the Pliocene Period. To bring a hitherto unknown ape into England in the Pleistocene Period involves an upheaval of palaeontological teaching, and on the present occasion adds an element of improbability to Mr. Lyne's contentions which is even greater than that which I have already indicated. And what is to be put on the other side of the balance against this enormous weight of improbability? Merely the possibility that there may be a few years'—perhaps not more than four or five—discrepancy between Mr. Lyne's suggestion of the age and mine, both based, not on absolute and constant data, but on the inference from notoriously variable factors in modern man, applied to the unknown conditions in an extremely early and primitive member of the human family. It seems to me that until some more positive evidence against the view is adduced, the only logical inference one is justified in drawing from the Piltdown fragments is that they all originally formed part of one individual. Although my conclusions are thus completely at variance with those which Mr. Courtney Lyne has set forth this evening, I am grateful to him for calling attention to a new aspect of the Piltdown problem, which is one of exceptional interest and importance.

MR. PYCRAFT (British Museum): I am afraid I cannot add anything to what has already been said by earlier speakers in the discussion. But I would suggest that a point—no doubt a small point—has been missed. Mr. Lyne has been dealing, so far, with human skulls having a microdont dentition; but the Piltdown man had a macrodont dentition. If the Piltdown tooth be compared with the canines of skulls such as I have in the British Museum—those of Torres Straits Islanders, Australians, or Tasmanians, whose teeth are much larger—I think a very different conclusion from that of the author would be arrived at. I do not think much can be based on his assumptions at present.

MR. MONTAGU F. HOPSON: I think it is due to Mr. Courtney Lyne that I should make a few remarks on his paper. He was good enough to submit it to me some months ago in rough draft, which had also been seen by certain other gentlemen present who have already spoken. It seemed to me to be essentially a paper which should be read before this Section of the Society, and so I persuaded him to offer it to us rather than to submit it to the Geological Society, seeing that the whole trend of his paper was distinctly dental, dealing with the macroscopical, and also to some extent with the microscopical, anatomy of this particular tooth. I must say I am a little disappointed with the discussion so far as it has gone, because I feel that the real aspects of the case, from the dental standpoint, have not been adequately met. Not that I, by any means, am sufficiently well qualified to deal with those particular

points. The chief argument I had in my mind against Mr. Lyne's suggestions with regard to this tooth was based on the extent of the wear shown by the tooth. That has already been explained—it shows an enormous amount of wear. But I recollected that I was shown—and I have already figured it elsewhere—a fragment of the mandible of a Neolithic child, apparently of the approximate age of 5 years, a specimen which was once in the possession of Mr. Mummery. The deciduous molars showed enormous attrition, and that, as Professor Elliot Smith has pointed out, might be attributable to the particular diet on which the child subsisted. But there were molars which—if we accept the age as being somewhere about 5 years, for the first permanent molars had not erupted—after three years of wear, showed a remarkable degree of attrition. Another point which occurred to me was this (and it has already been mentioned by Professor Elliot Smith): We can only presume what was the age of this particular individual, as of all types of prehistoric man; we cannot say definitely that the conditions which hold to-day for modern man with regard to the time of the eruption of the teeth or the closure of the sutures correspond with those of modern times. Personally, after considering the matter carefully, I am rather inclined to place this Piltdown man at an earlier age than 30—considerably earlier. And in doing that one remembers that the canine tooth in the anthropoids is the last tooth to erupt, and it would therefore be in a state in which we should find normally a large pulp chamber and pulp canal. There is another point in connexion with this Piltdown mandible which struck me when I examined it, and it was, that if you look at it from the lateral aspect you will find that the third molar is not free of the ascending ramus; a portion of the crown of the third molar, when it was present, must have been inside the ascending ramus, and that is a condition which frequently holds in modern people in the young jaw. But in the case of the Heidelberg mandible it is free—it stands clear away; you can see, when viewed sideways, the whole of the crown of the third molar. If you look at the Spy mandible, one of the most remarkable points is that it is free of the ascending ramus—and there is almost sufficient space to accommodate another tooth. In the Piltdown specimen it is obvious that the third molar was partly covered by the ascending ramus, and it may be that is a contributory point in the view that the mandible is younger than we might otherwise expect. I did not altogether agree with Mr. Lyne when he said the pulp cavities of the molars are normal compared with those of modern man. I was hoping that radiographs of them would have been thrown on the screen, as was promised, but they appear to me to be much larger than normal. If it could be accepted that this Piltdown mandible was a fairly young one, it might be that we should have a pulp cavity in the canine almost as large as that which Mr. Lyne has so much emphasized to-night as being out of the ordinary. In conclusion, I may be allowed to say I feel that I have, in a measure, done the Section some service in persuading Mr. Lyne to read his paper before us, and it seems to have been justified by the discussion which it has evoked.

Mr. J. G. TURNER: The question of attrition, which Professor Elliot Smith spoke about, is one which I have been watching. I have been examining a large number of Egyptian skulls, and one notices how quickly the first permanent molar is worn down. Within six years of its eruption it is quite flat, as flat as the first permanent molar of the Piltdown. But the second permanent molar takes considerably longer to become worn down, that is to say, the person goes on masticating on the more forward teeth, and the bicuspid will receive as much attrition as the second molar. And so in regard to the Piltdown tooth, finding the first and second permanent molars at one level and the cusps all gone, one begins to think it is a little older than some of us are suggesting at present. The point of the third molar is one which I noted some while ago, that it is not disimpacted. By the look of the jaw, I do not think we can argue youth on that account. The edge of the alveolus is—either by post-mortem or ante-mortem change—lower than it should be; it has been to some extent destroyed. If it be by ante-mortem change, one may argue that that person lived for a considerable time on a diet which induced a certain amount of stagnation around the necks of his molars: in fact, as has been pointed out by a Frenchman, there is slight evidence of pyorrhoea in almost every human skull, however ancient, which is known to us. At any rate, there is the fact that those alveolar borders are not perfect, and that argues the lapse of some time before that appearance would take place, if it be truly an ante-mortem change. As to the size of the pulp cavity, I have cut open a large number of teeth, with the specific object of examining the pulp cavity and the root canals. I did not come across widely open tooth canals in teeth of any considerable age, except once, in a man aged, perhaps, 35. The man's tooth broke so readily when I tried to take it out that I was led to examine it carefully, and I found the pulp canals widely open down to the apex, in a condition which absolutely surprised me. It was a two-rooted tooth, and I suppose there was as much uncalcified as calcified tissue in the roots. Another apex, which I think I still have in my possession, I put down as "query" as to whether it had been absorbed or whether it was the normal apex of that tooth, because it was so widely open; the apical opening ran up in a funnel shape to join the root canal. The tooth was a fully formed adult tooth. In our microdont dentition these are the only two cases I have found showing a large pulp cavity or root canal at an advanced age. For the rest, if there is anything in the point as to macrodont teeth, we shall have to go over the whole subject again.

Mr. COURTNEY LYNE (in reply): May I, Sir, thank the members of the Odontological Section of the Royal Society of Medicine for the kind way in which they have received my paper this evening? I realized when my paper was accepted that it would be a case of the cat among the pigeons; but the criticism to-night has, if it has been *fortiter in re*, also been distinctly *suaviter in modo*. I wish you to understand that the strongest argument which could have been brought against my views to-night would have been for Dr. Smith

Woodward, or Professor Underwood, or Mr. Pycraft, or Professor Elliot Smith, to have thrown on the screen a series of slides controverting my slides and showing conditions as seen in the Piltdown pulps. I am sorry if I misrepresented Professor Elliot Smith; now it certainly seems that he has thrown out an opportunity for reducing the age to much less than I had implied for him, and the matter becomes less important so far as he is concerned.

I will now deal with Professor Keith's view as to the wear on the tooth—not the *amount* of wear, but the *kind* of wear. In his reconstruction I think the wear is supposed to have been produced by the upper lateral incisor, mainly, at any rate. I suppose every one of us in this room is aware that we can protrude the lower jaw, and that animals can and do protrude it in that way when trying to attack or bite one another. I have tried to get the forward bite of his reconstruction within the last twenty-four hours, and find that the tips of the canines, when that is attempted, simply impinge on the first premolars, and will not allow the mouth to shut at all. This also seems to happen with Dr. Smith Woodward's reconstruction at South Kensington. If the jaw is protruded at all, the upper canine will prevent the full closure of the molar teeth or the incisors; the tip of the upper canine will come against the lower premolar. This arrangement is allowed for in anthropoids; you get the canines standing in a further-out line than the premolars, and that allows the upper canine to get a real clinch on the food or on the animal which it may be attacking. This condition is very much more marked in the Carnivora, where the anterior lower premolars are dwarfed, and the compression of the mandible in the lower premolar region allows the jaw to protrude and the upper canines to become consequently functional.

With regard to the question of the proportion of the tissues, I do not think anyone has answered that to-night. I threw on the screen Dr. Smith Woodward's own skiagram, which gives a picture of a certain proportion of enamel and of dentine. As far as my experience among Primate teeth is concerned, I find there is a rising proportion of enamel tissue as you approach the human species, and in this species you find a very much larger proportion of enamel than lower down in the Primate order. And here to-night you have a tooth which must be, according to the statement of these gentlemen, belonging to the *Hominidæ*, with a proportion of tissues which is, if anything, lower than adult living anthropoids display. If you look at the skiagram of the mandible you will find that the bony trabeculae are a much nearer approach to the Krapina man than those of the chimpanzee. In my opinion, the points which have shown Simian tendencies in this mandible have been magnified to the utmost possible extent, and the points which were typically human have only been brought in sufficiently to correlate, apparently, this mandible with the cranium. If you take the Galley Hill skull and look at the radiogram of the teeth, you will find there is a correlation of pulp cavities in the series.

With regard to the idea that the remains may belong to three different types of creature: a member, say, of the Primates, another portion belonging to a human being, and another part to a chimpanzee. I am not responsible

for these views. I admit the mandible goes with the cranium. Consequently, then, you have only the odd canine tooth to deal with, so far as I am concerned. Dr. Smith Woodward has told us, in a paper which he gave on "Dryopethicus"—and I draw attention to it—that the only evidence we have of anthropoid apes in Pliocene times in Europe is a few of their teeth.

With regard to Mr. Hopson's criticism on the subject of wear, I would have liked to have seen thrown on the screen to-night a picture showing anything like the amount of wear and tear (in a permanent tooth), such as the Piltdown tooth displays, and with a similar pulp cavity. You will find, when the wear is very heavy, that the pulp cavity is practically obliterated. And that is a point I would suggest to Dr. Smith Woodward when he deals with the Australian skull later on. It will be well to get an X-ray picture of the pulp of that canine, and also to show the proportion of dental tissues.

I thank Mr. Turner for his kindly criticism, and again I thank Mr. Hopson for bringing my paper before the Section.

Odontological Section.

President—Mr. H. BALDWIN.

(February 28, 1916.)

DISCUSSION ON WAR INJURIES OF THE JAW AND FACE.

The PRESIDENT (Mr. H. Baldwin).

By the term "war injuries of the jaws" we intend to imply those severe injuries of the maxillæ which are caused by projectiles such as bullets, pieces of shell, or bombs striking the bone at high velocity. The effect of these impacts is to comminute the bone and generally to destroy or completely carry away some portion of it. Pieces detached, and likewise teeth, frequently have so great a proportion of the momentum of the bullet imparted to them that they themselves act like projectiles and tear through the soft tissues in a radiating manner, inflicting very large flesh wounds.

As you know, the lower jaw is the most loosely attached bone in the body, though worked by four pairs of powerful muscles—the muscles of mastication. Therefore it happens that, when the bone is not only fractured but comminuted, the pull of the muscles on the remaining portions of jaw is unbalanced, and deviations of these fragments are the result, which deviations are intensified by the contraction of cicatricial tissue, and the case is further complicated by the formation of vicious adhesions if the jaw be improperly treated or not treated at all. The remaining teeth are thrown out of gear, and all the functions of the jaw are most seriously impaired. Facial deformity results, and the patient's life is made a misery by dribbling of saliva, obstruction of the breathing, and in many other ways. These things being so,

obviously the needs are, as far as possible or desirable, to regulate the remaining portions of the jaw—i.e., to restore them to their proper positions relative to each other, to retain them sufficiently rigidly, and to favour Nature's efforts at repair by every means in our power.

To effect these results, apparatus must be used inside the mouth. This apparatus should be as neat as possible, as mechanically perfect as possible in regard to its construction, and the principles of its action should offer no impediment to free irrigation of all parts of the mouth, should be as hygienically perfect as possible, and should occasion the patient as little discomfort as possible. The production and adjustment of such apparatus or splints as these are obviously outside the range of action of the general surgeon. The work is inevitably within the sphere of the dental surgeon or stomatologist. To demonstrate this and to add to our knowledge of the subject, we have organized the collection of dental splints, models, skiagrams, photographs, &c., now on exhibition in this building.

Being aware that the greatest amount of clinical material available to us was to be seen in France, several of my colleagues and I have been over to France somewhat recently to study the conditions under which these cases were being treated and the methods adopted there. We found that organization of the treatment had been rapidly developed during the progress of the War, and that now all these cases are being assembled in special hospitals under the care of specialists; that no time was now lost in sending men wounded in the jaws direct from the Front to special hospitals, and that all the old cases which had been aimlessly placed in other hospitals, and therefore neglected as regards jaw treatment, were now withdrawn from them and collected in special hospitals. Various cities—viz., Paris, Lyons, Bordeaux, Marseilles, Bourges, and Clermont—are now made stomatological centres.

We visited Paris and Lyons, and found in each of these cities admirable organization of the jaw treatment, and invariably a dental surgeon at the head of each establishment. In Paris the jaw cases were divided between the Dental Department of the American Ambulance under Dr. G. B. Hayes; the Military Hospital of Val de Grâce, which has one wing devoted to jaw cases under Surgeon-Major Leon Frey, his department being entitled "Service de Stomatologie et de Prothèse Maxillo-faciale"; the École Dentaire de Paris, coupled with the Michelet Hospital under Dr. Maurice Roy, and a private hospital of the Comtesse de Grammont, conducted by Dr. Hotz—aggregating about 1,000 beds

for jaw cases. I have much pleasure in announcing that Dr. Hayes and Dr. Hotz have done us the honour of coming over from Paris on purpose to be present at this meeting, and have both contributed very valuable material to the exhibition.

Dr. Roy has sent a remarkable collection of photographs, and Dr. Frey had kindly promised to send us what he called "un petit travail," but greatly regrets not having been able to get it ready, through being sent away on Government business.

At Lyons we found a wonderful organization under Dr. A. Pont, with 830 jaw cases assembled in six large hospitals, the central institution being installed in a large school building, and called the "Hôpital de Stomatologie et Prothèse Bucco-faciale." There are five other hospitals annexed to this central hospital. One is reserved for jaw injuries complicated by wounds of the sinuses and ear, which are under the care of an experienced oto-rhino-laryngologist as well as the stomatologist. Another is reserved for jaw injuries complicated by wounds of the eyes, and has a regular service of ophthalmology. Dr. Pont not only directs this vast organization, but himself does all the important surgical operations such as osteotomies, removal of missiles, insertion of adipose grafts, bone-grafts, &c., as well as all the plastic operations for mending the tissues of the face and neck which these cases may require.

Dr. Pont has contributed to our exhibition an album of photographs of cases before and after treatment and a typewritten illustrated monograph on the methods employed. He is mobilized and under orders, otherwise he would also have honoured us with his presence at this meeting.

I will here take the opportunity of most cordially thanking these heads of the Service of Stomatology whom we met in France for their unfailing cordiality, courtesy, and kindness. They were, one and all, remarkably kind, and spared no pains to be of the greatest possible assistance to us.

In the early days of the War Dr. Pont started an ambulance of thirty beds for face wounds. It began work on September 15, 1914. In November, 1914, the War Ministry created Paris, Lyons, and Bordeaux as stomatological centres; Marseilles, Bourges, and Clermont have since been added. Dr. Hayes, in his report dated September 1, 1915, gives the number of his staff as thirty-two persons—including eight dental surgeons, eight dental mechanics, three apprentices, two nurses, seven auxiliary nurses, and a typist. The ambulance contains

560 beds, and in May, 1915, 125 of these were occupied by jaw cases. In addition, the dental department was treating its own convalescent cases, who had gone out and were residing in eleven other hospitals. The construction of all the dental splints, other apparatus, and dentures was only possible by the assistance of the trained dental mechanics; and Dr. Hayes congratulates himself on the advantages gained by their being mobilized for this purpose. The work brought the general surgeons and dental surgeons into co-operation and interdependence, and they always worked most harmoniously. Almost every case presented fractures with loss of substance, sometimes amounting to nearly the whole jaw or half the face. Means were devised to maintain fragments in their normal relation during healing, and later on lost parts were restored, and a framework was arranged to support the bone-grafts or whatever plastic operations the surgeons decided upon. Thus it was possible to return to the world not mutilated wrecks, objects of horror and commiseration as they had come in, but men presentable and happy.

The want of a stomatological service early in the War resulted in old cases coming to the hospital, their mouths sewn up, faces distorted, mastication impossible, speech very difficult, fractured jaws ununited or displaced, ossified, or ankylosed in wrong positions, the work of restoration being doubled, and requiring either surgical operation or long and tedious treatment for regulation by means of complicated mechanical apparatus.

In our own country, at Norbury, near Croydon, there exists one military hospital specially set apart for jaw cases, which is excellently conducted by Mr. J. F. Colyer as dental surgeon or stomatologist, and Captain Holt, the local R.A.M.C. commandant, who, fortunately, cordially supported Mr. Colyer in his endeavour to make the hospital a stomatological centre.

It would not be in excess of the requirements if a special hospital were established in the district of each Command in the United Kingdom. In my opinion, our own Army dental and stomatological arrangements have always suffered from the lack of an experienced and competent officer on the staff of the Director-General, to advise on important matters in connexion with dental and stomatological work, to help in the organization of dental service at home and abroad, to be responsible to a certain extent for the execution of plans and details. In Australia and New Zealand such dental officers have been recently appointed.

Two Fellows of this Society and members of this Section who have always practised dental surgery before the War are Lieutenants-Colonel in the Royal Army Medical Corps. They are both known as efficient organizers, and are eminent as teachers of dentistry, with long hospital experience. During the War they have distinguished themselves, one at least being mentioned in despatches for distinction. Their services in the War, however, have been used in an ordinary medical capacity and not at all in a dental capacity. I suggest that one of them might have been even more useful on the War Office Staff as a Principal Dental Officer.

The French War Minister is just now introducing a Bill to the Chamber of Deputies to extend widely the use of dentists in the Army, and to assure to them a rank and duty proportionate to their professional capacity and scientific knowledge.

I am sure this Society, with its knowledge of the personnel of the profession, would place itself disinterestedly at the service of the War Office, if desired, in any efforts that might be made to establish special hospitals for jaw injuries in London and in each Command.

I venture to suggest that all is not being done in this country which should be done for soldiers wounded in the jaws and face, and in order to assist matters I also venture to suggest that a care committee for soldiers wounded in the jaws and face should be formed, the object of which committee would be to promote the interests of such cases not only while still in the Army but also after their discharge, when it would arrange that they should go on receiving equally skilful expert attention at special hospitals as before. The nucleus of such a committee has been formed, and it is to be hoped it will receive influential additions and support.

MR. J. LEWIN PAYNE.

In response to the request of the President and the Council of this Section, I have the honour of opening the discussion upon the subject of war injuries of the jaws. Personally, I regret that a member of the Section with greater knowledge and larger experience, who could have spoken with authority, has not undertaken the opening of this debate. I must add that even if I were competent to undertake a complete survey of the whole subject, I should consider it undesirable in the presence of many members well qualified to

contribute to a discussion in all its aspects on this subject. Moreover, the President has assured me that a comprehensive survey is not required by one speaker, and that it is desired that each contributor should rather give his individual experience of the cases he has seen and of those that he has been called upon to treat. I propose, therefore, to deal briefly with the types of cases met with, the nature of the displacements, the general lines of treatment, and to show lantern slides of a few typical cases that have come under my care.

My personal experience of war injuries of the jaws is based, first, upon some twenty-two patients who have come to me for treatment. In addition to these patients I visited France on two occasions, in August and December, 1915. At each of these visits large numbers of soldiers suffering from every description of jaw injury were seen under treatment. The dental surgeons of France have always been noted for their skill in this branch of surgical prosthesis, and it would be difficult to exaggerate the completeness of the organization and the benefits which are being afforded to the wounded soldiers of that country, and, indeed, indirectly to all humanity, by the work which is being carried on in various hospitals of France. The President has already referred to this, and I will only add the expression of my own sincere thanks to our colleagues there for the kindness and help received in the course of the visits of the past nine months. I would like to add that it has been my privilege also to see a number of patients at the Croydon and Wandsworth Military Hospitals, where such excellent work has been accomplished in this direction.

In discussing war injuries of the jaws we are considering those severe fractures of the mandible and maxillæ arising from rifle bullet, shell, or bomb wounds. Such injuries differ from the ordinary fractures met with in civil life in the frequency of multiple fractures, the comminution of bone, the loss of substance, the degree of displacement of the fragments, the frequent presence of foreign bodies, such as pieces of teeth, bone, bullet, or denture; and also in the cicatrization of the soft tissues, which frequently leads to the elimination of the buccal sulcus. To these injuries may be added the complication with other wounds of the face, and the relatively greater frequency of injury to the superior maxilla. The chief trouble is nearly always in connexion with the mandible, which is recognized as being the most used bone in the body. For it is not only more liable to serious damage, in consequence of its exposed position, its loose attachment to the skull, and the action of powerful muscles, but displacements also tend to be

relatively greater than in other bones of the body. The mere use of bandages and other external appliances seldom avails to correct the deformity. The question has been asked: Is it possible to divide these injuries into a number of typical cases? In reply, it has been suggested that they might be grouped into old and new cases, the old being those of long standing and those in which the bones have already become united, but in mal-position. Other divisions might be: simple fractures and multiple fractures; or, again, fractures with, and those without, loss of substance. Generally, a classification denoting the clinical aspects of the injuries seems to present a more complete grouping, and from observations made upon cases investigated I should suggest a division into the six following types:—

(1) Fractures of the mandible, without displacement of the line of occlusion.

(2) Single fractures of the mandible with lateral displacement.

(3) Single fractures of the mandible with vertical displacement.

(4) Two or more fractures of the mandible with loss of substance.

(5) Gun-shot wounds of the maxillæ.

(6) Fractures involving loss of the anterior portion of the mandible, the maxillæ, or of the whole of one side, together with the soft tissues adjacent.

The fractures are so variable in character that it is difficult to include every form without making a long list, but I believe these six types will embrace almost every case.

Fractures of the jaw in war-time are usually accompanied by loss of bony substance, and when this occurs they almost invariably present some type of deformity, but in a certain small number of cases of gun-shot wounds of the mandible there may be found no disarrangement in the line of the teeth, even though there may be comminution of bone. These cases may be grouped under *Type 1*. Fig. 1 shows the models of such a case. In the skiagram (fig. 2) the comminution of bone is observable.

Type 2: Single Fractures of the Mandible with Lateral Displacement.—The wound is usually between the mental foramen and the angle. There is loss of substance, the jaw is pulled over towards the injured side, and the occlusal surfaces of the teeth lose contact with the antagonizing teeth, the function of articulation being no longer possible. The fragments of the jaw tend to become displaced into the space caused by the lesion. Fig. 3 illustrates lateral displacement caused by a fracture, with loss of substance, at the angle of the mandible on the right side.

Deformities of Type 3: Single Fractures of the Mandible with Vertical Displacement.—These are commonly found in the incisor region and in situations where, in consequence of the obliquity of the fracture and the pull of the muscles, one fragment tends to slide above

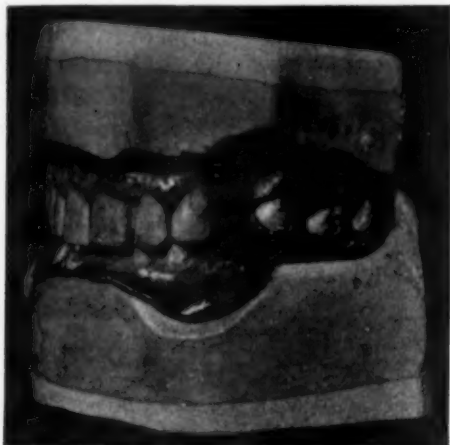


FIG. 1.

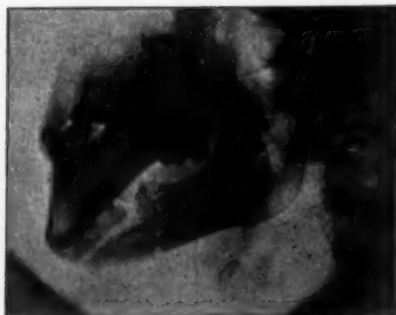


FIG. 2.

the other. Such vertical displacement is seen in fig. 4. There was no loss of substance in this case.

Type 4: Two or more Fractures of the Mandible with Loss of Substance.—When the mandible has been fractured by a bullet or shell

wound on both sides, the anterior fragment will be drawn backwards if there has been considerable loss of substance involving the basilar border. In other cases the anterior fragments fall forwards, and there is usually a considerable separation between the upper and lower incisors. The posterior fragments are drawn together towards the middle line, and if the fracture involves the molar region the angle of

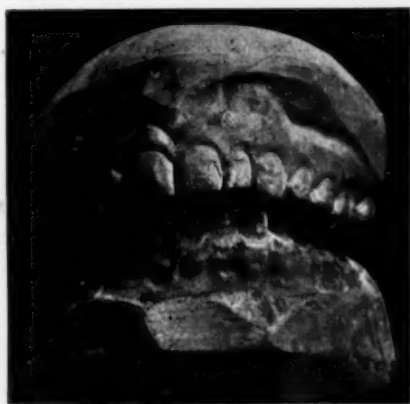


FIG. 3.



FIG. 4.

the jaw will tend to be tilted upwards. Fig. 5 shows the common deformity met with in cases of Type 4 when there has been loss of substance on both sides down to the basilar border. The radiogram of the same case is seen in fig. 6. Fig. 8 shows the models of another patient's mandible with deformity of Type 4. Fig. 9 is the same case

as shown in fig. 8, with the models articulated, before the dental treatment had been started.

Type 5: Gunshot Wounds of the Maxillæ.—Maxillary fractures, though more simple to treat from the prosthetic standpoint, may give rise to troublesome suppuration from the antrum becoming involved, or from some tooth or missile being buried deep within the bone. The cranial bones are often fractured by the same injury, and the risk of subsequent hæmorrhage must be remembered. Fig. 10 is a radiogram of the fractured maxilla, showing the root of a premolar buried deep in the substance of the bone.



FIG. 5.

Type 6: Where the Anterior Portion or more of the Bone and Soft Tissues have been carried away.—These are the most severe and distressing cases. The appearance of the patient is ghastly, mastication impossible, speech very difficult, and when the chin and symphysis have been shot away there is no control of the saliva. Fig. 11 shows the side and front face view of a patient who had lost all the anterior portion of his maxillæ from the second molar on the left side to the second premolar on the right. The upper lip and lower portion of the nose had been shot away, and there was a fracture of the mandible on the left side with loss of substance. Fig. 12 shows the face mask and model of the upper portion of the mouth of the case shown in fig. 11.

It should be noted that the greatest destruction is nearly always found at the point of exit of the bullet. Trismus following fracture of the mandible is common in cases where the ascending ramus or the



FIG. 6.

Radiogram by Dr. A. C. Jordan.

condyle is involved. There may be no mal-occlusion, but there will be loss of function.

In the main principles of the treatment of jaw injuries there is



FIG. 7.

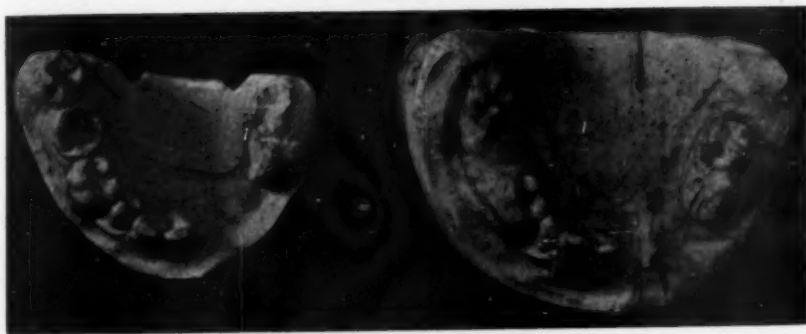


FIG. 8.

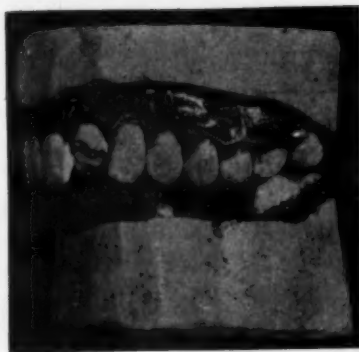


FIG. 9.

probably general agreement amongst dental surgeons, but it will not be surprising if the methods employed are as varied as the number of men that have undertaken this work, for, to a large extent, we are breaking new ground. In answer to the question, At what period after the injury should the aid of the dental surgeon be called in? I would say emphatically that it should be as soon as possible after the wound has been inflicted. Even if it be not possible to apply a splint or



FIG. 10.

Radiogram by Dr. H. A. Eccles.

other appliance at so early a period, the dental surgeon will be able to attend to the cleanliness of the mouth. It should certainly be his duty to decide the best time for the fitting of an appliance. The general surgeon does not always appreciate how much assistance can be given to him by the dental surgeon in the restoration of the soft tissues; yet it is essential that there should be close collaboration

between them. A lack of this co-operation often leads to operations of unnecessary severity, involving the destruction of living tissues which might have been saved. One has seen cases in which useful teeth with live pulps have been extracted, whilst septic roots have been allowed to remain. In other instances the soft parts have been sewn up around bony fragments which were still displaced; this has led to cicatricial contraction of the soft tissues, which only complicates the trouble when the treatment of the jaw is undertaken. No attempt should be made to perform a plastic operation for the purpose of closing the gap until the interdental splint has been placed into position. The dental surgeon should begin his work immediately after the patient has recovered from the condition of shock. Such promptitude will save much of the restoration to occlusion that is demanded in neglected cases. If this



FIG. 11.

aid for any reason, however, is not available, these cases should be transferred as soon as possible to a hospital where it is always at hand.

Efficient drainage, as in all septic wounds, is the first essential principle of treatment. Thorough irrigation of the parts must be carried out from the start. Some simple antiseptic, such as Wright's saline solution, should be used from an elevated tank or a spray, and in severe cases this must be repeated by the nurse every few hours. Skiagrams should be taken at the commencement, during, and at the conclusion of the treatment; for these will not only aid materially in the diagnosis, but will indicate also the progress and success of the treatment. The dental surgeon must see that the teeth in the mouth are freed from tartar, blood, and food. Carious teeth should be temporarily filled. Septic roots and teeth which cannot be rendered clean should be extracted

as soon as possible; and any tooth that is situated directly in the line of the fracture usually has to be sacrificed, or union may be delayed. The application of the splint should follow as soon as it can be borne by the patient. Often it is desirable to use some temporary appliance which can be replaced by the permanent splint when the conditions become more favourable.

Wounds of the maxillæ usually present small difficulty in regard to restoration as compared with wounds of the mandible, because of the attachment of these bones to the cranium. The maxillæ have



FIG. 12.

the advantage of the support of other surrounding bones and of a free blood supply. In most of these cases displacement is easily corrected, and only requires holding in position by simple bandages or by means of a suitable appliance. Support may be obtained from the mandible when there is a tendency for the splint to move, or, if the mandible also has been fractured, by extending the maxillary splint outside the mouth and obtaining stability from straps passing over the top of the head. It is often possible in the upper jaw to save loose teeth that in cases of similar damage to the lower jaw would be lost. If sepsis can be

avoided union usually occurs in a few weeks, but the chief troubles arise from the opening up of sinuses or from hæmorrhage.

When sepsis has been controlled by constant irrigation and by the removal of all sources of irritation such as the bullet, pieces of shell, septic roots, and dead bone, the next important step in the treatment is to restore what may remain of the masticatory apparatus by bringing back the fragments of the jaws into a position which permits of normal occlusion. As Surgeon-Major Leon Frey, of Paris, has so well explained in the *Paris Médical* of August 15, 1915, the restoration treatment may be divided into four stages:—

- (1) Reduction of the displacement of the bony fragments.
- (2) Retention of these fragments in the position which allows of normal occlusion.
- (3) Reduction of cicatricial contraction, the restoration of muscular equilibrium, and the remoulding of the facial contours.
- (4) Fitting of a permanent prosthetic appliance.

It will not be possible now to give even an outline of every type of appliance that is used for the correction and restoration of jaw deformities, but it may be said in general that the more simple the appliance the better, so long as it is clean and efficient. Some splendid examples of the work which can be accomplished with simple apparatus have been shown by Dr. Hotz in his specimens in the museum. The aim of prosthetic treatment should be:—

- (1) To correct the deformity and restore the teeth to the position of normal occlusion.
- (2) To retain the fragments in this position until union has occurred. These appliances should allow modified movements of the jaw to occur. Rest, not absolute fixation, is what is required; in fact, union will occur sooner if passive movements of the mandible are allowed.
- (3) Later, some form of denture may have to be fitted to make good the gaps left by lost bone and teeth, and to restore the functional activity of the jaws. The appliances employed for these objects may be divided into three groups by utilizing terms which have been suggested in separate papers by Surgeon-Major Frey and Mr. Stanley Mummery:
(a) Inter-fragmental, (b) inter-maxillary, (c) cranio-maxillary.

(a) The *inter-fragmental* consists of splints depending for their grip and action upon the jaw they are intended to correct.

(b) The *inter-maxillary* are those appliances in the employment of which portions of both the mandible and the maxillæ are used in the

correction of the deformity. The anchorage is based upon one jaw and the force is exerted upon the other.

(c) The *cranio-maxillary* type of apparatus, as employed in cases when teeth are absent in both jaws, or when the teeth are too diseased to support the bands, ligatures, or flanges of an inter-maxillary apparatus, or in cases where both jaws have been fractured.

It has been suggested that the employment of an interdental splint at an early stage tends to promote sepsis. This is an entirely erroneous idea. Splints, properly constructed, should not only be easily kept clean, but should reduce the period of suppuration by arresting the mobility of the fragments, and by assisting the irrigation of the parts. Also, it must be remembered that a well-fitting splint very considerably reduces the discomfort of the patient. Moreover, the use of mechanical restoration appliances, as soon as possible after the infliction of the wound, will prevent the cicatricial tissue from pulling the remaining portions of the jaw out of their normal place during the process of healing. During the third stage in the treatment frequent massage, both manual and electrical, by competent nurses should be adopted to restore muscular equilibrium, and care should be taken to guard against cicatricial contraction. Fortunately, cicatricial tissue is peculiarly responsive to pressure. An appliance with gradually increasing pressure may be employed to stretch the cicatricial band, and in this manner to restore a lost sulcus.

To sum up, the conditions favourable to osseous union are :—

- (1) Rest for the fragments.
- (2) The removal of foreign bodies from the region of the lesion.
- (3) Cleanliness of the mouth and its surroundings.
- (4) Massage.

The subject of bone-grafting and bone-plating I must leave to those who have had more personal experience in this treatment. I will only say that of the six or eight cases of bone-grafting in the mandible that I have seen the grafts taken from the tibia seemed to be the more successful.

The time has not arrived for dogmatic statements in regard to the details and methods of treatment of these war injuries of the jaws, and doubtless there will be differences of opinion upon the views which have been expressed in this paper. Nevertheless, I would say, in conclusion, that I believe there will be general agreement amongst dental surgeons upon these four points:—

(1) That nearly all the jaw injuries of the War require some dental treatment.

(2) That the dental treatment should be commenced as early as possible.

(3) That the patients should be referred to dental surgeons who have made themselves familiar with the treatment of jaw injuries.

(4) That it would be greatly to the advantage both of the patient and the surgeon if all of these cases were segregated in special hospitals devoted to this class of work.

Dr. GEORGE B. HAYES.¹

I bring to you greetings of the Dental Staff of the American Ambulance at Neuilly, and assurances of their hearty support in your efforts towards the establishment of proper lines of treatment and proper organization of service in this field of work, which has brought the general surgeon and the dental surgeon into a far more intimate and interdependent relation than ever before, and has demonstrated not only the advantages to be gained, but the necessity of their mutual consideration and co-operation. It is a privilege and honour to be present on this occasion, and to take part in a discussion by such a distinguished body of men. As a result of our efforts I have brought with me thirty cases out of 380, which have been treated by my staff, with the hope that from our work and experience some benefit, slight as it may be, may accrue to those of you who have not yet had the advantage of extensive treatment of these cases.

From visits to various hospitals, and from reports distributed, it would seem that successful results are being obtained by means and methods wholly different, dependent upon individual ingenuity and upon a particular disposition or preference for certain kinds of appliances, but all necessarily aiming towards the restoration of the injured parts to normal forms and functions. No new pathological problems or conditions have been met with. The difficulties encountered are due to the variety and complexity of conditions presented, which demand most careful consideration. The presence or absence of a single tooth on a fractured portion may suffice entirely to change the method of treatment, while other factors such as trismus, ankylosis, partial con-

¹ Chief Dental Surgeon at the American Ambulance at Neuilly, France.

solidation, the presence of sequestra, foreign bodies, and cicatricial adhesions, necessarily influence the immediate course of procedure and determine the final method of treatment.

These problems are being successfully solved by skilful men in the various centres in France, where special departments have been organized for these cases, and a list recently obtained from the Department of Health (*la Santé*) mentions twenty-nine such centres.

From personal experience the greatest advantages seem to obtain where these cases are centred in a general hospital, uniting under the same direction the different specialists called upon for assistance, and especially is this true owing to the frequent complications arising from additional wounds of the body and extremities necessarily retarding the period of convalescence or the moment when the patient might be transferred to a special clinic.

The principal treatment in these cases falls upon the dental surgeon, and the ultimate success depends upon the building up and restoration of the jaws as a skeleton or framework upon which the final plastic operation is rendered possible. This fact became evident a few months after the beginning of the War by the arrival of cases which had come into hospitals without a dental surgeon. They had passed beyond the general surgeon's help. They were cured of infection, their wounds had healed. Many had received a finishing plastic operation, but the buccal orifice had been so diminished that it was impossible to make a thorough examination of the mouth or to introduce an impression tray. They came with faces distorted, mastication rendered impossible, fractured parts not only displaced but consolidated, necessitating for the reduction of the fractures either a radical surgical operation or a long and tedious treatment, for which some special apparatus had to be made.

The French were not long in creating special centres for the care of these cases, and it is of importance to note that in the recent order to evacuate such wounded as could walk (in preparation for an expected influx of other wounded) the jaw cases were allowed to remain, unless removed to a hospital similarly equipped.

Much has been done, but much remains to be done to supplement the special centres already created. There should be dental surgeons at the front to give first-aid in these cases, to decide what remaining fragments of jaws and teeth should be saved and what removed. They should be equipped for temporarily wiring the fragments, for the making

of simple splints. They should see to the proper bandaging when indicated, and prevent indifferent or contra-indicated bandaging, which in many cases only tends to increase the deformity.

The subject of war injuries of the jaw includes so much, that it is difficult to know where to begin and where to end. Many questions involving the treatment of these wounds are undergoing renewed and general study—any one of which might alone take up the time of this meeting:—

- (1) The choice of disinfectants and methods of application.
- (2) Bandages and contra-indications.
- (3) The making of interdental splints, together with a study of cases requiring—
 - (4) Fixation of the mandible.
 - (5) Indications for the extraction of certain teeth.
 - (6) Means of overcoming trismus and its treatment.
 - (7) Indications for bone-grafting and choice of tissue to be grafted.
 - (8) The relative advantages of different kinds of force and means of application in the reduction of fractures.
- (9) A proper classification of these cases in reference to the line of treatment to be followed, with definite stages of operation.

Much of this work is new, and the best means and methods have yet to be determined, when careful statistics shall have been compiled, but from the great number of cases, from the interest aroused, and from the favourable conditions under which the various branches of surgery have so happily been united—especially in this Society—much is to be expected, and we look forward to the establishment of definite lines of treatment between the general and dental surgeon as to priority of operation, especially in cases involving extensive loss of soft tissue, cases where an immediate closing of the wound would greatly multiply the difficulties subsequently to be overcome in reducing the fracture, or where the delay necessary for the adjustment and fixation of an apparatus to prevent displacement and vicious union would render a final plastic operation less satisfactory through the formation of scar tissues and great cicatricial contraction.

Dr. Hotz.

I am invited by your President to say a few words on general principles in the treatment of jaw fractures produced by bullets or shell splinters. First of all, may I venture the opinion that in all jaw cases combined with an extensive destruction of the surrounding tissues, the general surgeon should call the dental surgeon into consultation, and perhaps take his advice before undertaking any plastic facial restoration. This co-operation would be beneficial to the wounded and save many of them from unnecessary and prolonged pain. The facial restoration would be much easier and more rapidly accomplished if the fractured maxillæ were first restored to their normal position; for if the surgeon intervenes first, the correct plastic restoration becomes almost impossible from an æsthetic point of view, and the re-establishment of the normal occlusion is postponed for a long time, thus allowing the fragments of the maxillæ to ossify in a faulty position. This requires surgical intervention afresh in order to enable the dentist to establish normal occlusion. Professor Tuffier, the surgeon in charge of Ambulance 39, in Paris, who did me the honour of asking me to take charge of the jaw cases, proposed that I should undertake the treatment of the jaw fractures, and only seek his aid when I thought that the conditions were favourable for his intervention.

The object of the dental surgeon in the treatment of jaw fractures is to re-establish normal occlusion and restore the masticatory functions. At the present time we are all familiar with the various appliances and splints that are employed to accomplish this. Every practitioner has his preference and uses his personal judgment with a view to obtaining the result he aims at.

I am in favour of simple rubber splints in most cases, with the exception of those few in which a steady maintenance of the fragments is indicated. Such an indication is the case in which some of the masticatory muscles have been severely bruised or cut, and, consequently, the remaining muscles pull the fragments up, down, in or outwards to such an extent that the splint has to be wired or cemented to keep the parts steadily together for a certain length of time. I object altogether to intermaxillary wiring, as this creates the risk of an ankylosis being set up. The use of movable rubber or metal splints permits of pressure being applied to the maxillæ as

well as to the teeth; this is a very important factor when great pressure is necessary to move the fragments into their proper place. The remaining teeth, especially those near the fracture, are generally loosened, and will easily yield to pressure. In fact, many of the teeth situated as above mentioned exfoliate in within a month or two, or sometimes they remain painful, produce irritation of the gums, and finally have to be removed. Then we discover that the roots have been fractured in their alveoli. In many cases we have to use pressure from without—over the chin or the cheeks. For this purpose we may employ linen bandages, or manufacture special chin cups of rubber or metal with hooks fastened to them for the attachment of elastic bands, that will allow of a pull in the direction indicated by the case.

I need not go into further detail respecting this question, so I will touch quite briefly upon another very important item—namely, the hygiene of the wounds of the oral cavity. We have all observed that when the wounded come from the Front their mouths are in a terribly septic condition. The pernicious odour emanating from the oral cavity is simply unbearable, and must be got rid of rapidly. I believe a good method of effecting this is to suspend a recipient fitted with a rubber tube and a glass cannula above the head of the patient; to fill it with a light tepid solution of iodine (from 5 to 10 per cent.) or chloride of sodium, and to wash out the oral cavity thoroughly from three to six times a day according to the septic condition present. The improvement that results is simply wonderful if the nurse follows the directions correctly: in two days the fever abates and the fœtid odour disappears entirely. We may then approach the patient for a thorough examination and begin the correction of the fractures.

MR. J. F. COLYER.

My contribution to the discussion this evening is purely from the practical point of view. I want to describe to you briefly the result of experiences in connexion with the Croydon War Hospital.

Croydon War Hospital has five divisions, and embraces about 1,000 beds. It is a hospital under the Eastern Command, and into it special cases are brought; for example, cases of nerve injuries, joint injuries, ear diseases, and cases of gunshot wounds of the jaw. So far, we have had something like 200 cases through our hands, and at the present

time we have in that hospital close upon 130 patients. I may remark at the outset that up to the present time we have dealt with practically nothing but ununited fractures. Many of the patients whom we have had to deal with have been injured six months, or even a year. I do not regret that because, as a matter of fact, owing to mistakes in treatment which we have seen we have learned how treatment should be carried out in future cases.

There are one or two points in connexion with the routine treatment about which I should like to speak. As soon as a patient is admitted into the hospital a peroxide of hydrogen mouth-wash is used every two or three hours, and a 2 per cent. solution of iodine is applied to the gums once every day. Skiagrams are obtained and short notes made. The patient is then taken to the operating theatre and given a general anæsthetic, usually chloroform. We then ascertain how far we can reduce the various fractures. The septic teeth are removed, as well as the teeth on either side of the fracture. That is a universal rule throughout the hospital, and those of you who have seen the work of the hospital will realize the immense practical importance of that measure. If that were done in the case of all patients with war injuries of the jaws, the country would be saved thousands of pounds.

The next step consists in the adaptation of splints; these are not necessarily fixed at the time—we often leave the fixation for two or three days. In many cases splints are allowed to remain removable, for I am one of those who believe that some degree of mobility in certain fractures is useful. Still more important, we never think of fixing the splints until we are quite certain we have got rid of all sources of sepsis. When the patient is practically convalescent, and the fracture has completely healed, a retention splint is inserted.

There arises here another very important practical point. We are dealing with a military question, and we are there to make these men efficient. If we return these men straight away to their depôts, they go sick because they are unable to masticate their food. Therefore they are now being put through a dietetic course before leaving the hospital: we pass from a fluid diet to a minced diet, then to a boiled diet, and, lastly, to a roast diet. So we are now turning men out to their depôts efficient so far as their masticatory powers are concerned. I am fortunate in having as my commanding officer Captain Holt, who is heart and soul in sympathy with the work; and when I suggested this course to him he set out with all his energy to make it a success,

and this was a matter of some difficulty, as it must be remembered that the diets had to be arranged within the military standard. Another advantage of bringing together cases of the character we are discussing to-night is in connexion with the nursing, for in jaw cases this involves special knowledge. I am pleased to say that at the Croydon War Hospital the nursing is carried out in a most efficient manner; there is a spirit of enthusiasm pervading the nursing staff, and the happy results we have had are in a great measure due to excellent nursing.

A point of some importance, also, is that the man with a fractured jaw is rather a hideous sight, and if he is in a ward with miscellaneous cases he is apt to be very self-conscious and bashful, feeling, perhaps, that life is not worth living. But if he is with other men who have been similarly injured, he finds that life is worth living.

I will now pass to discuss the question of certain displacements. Many of the cases of fracture in the region of the incisor teeth which look serious are not so serious. If there is a slight fracture of the lower part of the jaw, the first thing which happens is that the lower part of the mandible engages, and there is a tilt inwards of the fractured portion. There may be, apparently, a considerable amount of deformity, but that is easily overcome. If a shot has gone completely through, carrying away the mandible vertically between the canines, two points engage together, and there is a parrot-shaped mouth and loss of chin.

The most important displacement which we have to consider in the treatment of jaw injuries is that which occurs when there is a fracture in the region of the last molar tooth. In all the cases in civilian practice, that posterior fragment usually moves upwards and inwards. But in many of these war cases the impact of the shot drives the larger fragment inwards and the posterior fragment moves forwards and outwards.

Most of the fractures which we have to deal with in these cases involve loss of tissue; there is often a loss of tissue to the extent of $\frac{1}{2}$ in. or $\frac{3}{4}$ in., and a very large proportion of the cases have double fractures. The problem as to how to treat these is a very difficult one. If you take an ordinary case, what you find is that the posterior fragment is held up in position by its occlusion with the upper teeth and is unable to move forward, whereas the anterior fragment slews round to the injured side. If the anterior fragment is brought into correct position a gap remains between the fragments and only fibrous union will occur, but if the anterior fragment is kept in place and the molar teeth on the

maxilla opposing the lesser fragment are removed, this fragment will swing forward and osseous union will take place. Among the men who are here for your inspection I will show you some in whom this has happened. It is a very important point in the treatment of these fractures.

I shall not speak at any length about splints, because the splints which are used in the Croydon War Hospital are simple in the extreme. But there is one type to which I would like to refer, and that is the type which one might call a skull-and-mandible splint. It consists of a metal splint moulded to the outside of the mandible in a knitted skull-cap. These splints can be easily made out of aluminium by anybody, and the caps can be knitted by the ladies of the district. In Croydon we have a most ingenious ally in the Recorder of the Borough; he not only makes these splints for us, but devises many other things. In all these cases an outside splint covering the mandible is of the greatest comfort to the men. We do not use this splint for keeping the fracture in position, but rather to give rest and support to the mandible.

Another point is the cause of fractures being ununited. Obviously the cases of ununited fracture can be grouped as respectively due to two causes: (1) The presence of a foreign body between the fragments; (2) want of rest.

The main reason of an ununited fracture is the presence of the teeth in the line of the fracture; they practically act as a foreign body, and are a source of sepsis and prevent healing. Time after time we have had men admitted with ununited fracture of six or eight months' duration, and on removal of the teeth the fractures have got well in about three or four weeks. You will therefore realize why I mention that in these fractures the removal of those teeth should be undertaken straight away. Another very important point is to make sure you have not a foreign body between the fragments. At the Croydon Hospital we have not yet a special radiographic apparatus, such as we have in dental hospitals, and we have been at some loss over the absence of it; but I ask my radiographer to tell me whether there is a foreign body between the fragments. Once or twice the radiogram showed no foreign body and we have put up the fracture, only to find, after six weeks, that a foreign body has appeared. That has been taken out and healing occurred. Every foreign body between fragments which is missed means a delay of six weeks.

The other of the two points I mentioned is the want of rest. Many

of these patients are sent in having a see-saw movement; that is to say, the posterior fragment is upwards and the anterior fragment downwards; and every time the man tries to close his jaw, down goes the posterior fragment until the teeth in the anterior fragment are in occlusion. We have had many instances of this kind, and we find that directly we give rest to the fracture by removing the upper teeth these cases get well rapidly.

The next point is, that sinuses in connexion with fractured jaws are due to some cause, and in the majority of cases a septic tooth is the culprit.

A very important problem in ununited fracture concerns fractures in malposition. That includes a large part of the work we have to do. We see many cases in which the anterior fragment has slewed across. I have found by experience that the best way to treat these is by means of "stretching plates." We make a splint to the upper jaw and one to the lower teeth, and by a hook we drag the mandible as far across to the normal as we can, and adjust the two splints in position and then unite them. The splint is put into place, and the mandible drawn forcibly into place, and in about twenty-four hours, the soft tissue having stretched, the mandible engages easily into the splint. In this position it is allowed to rest three or four days, and is then pulled further into position. When we have pulled the mandible to the correct position the splint is fixed.

As practical persons, it is very important for us to discuss how far it is advisable to obtain an ideal jaw. I am sceptical as to whether it is right always to attempt this. The general principles which I have set for my own guidance have been: (1) The lives of these patients have to be saved, and no dental treatment can be carried out until this has been achieved; (2) Next I aim at getting osseous union, and if I can get it with good occlusion well and good. But if I risk losing osseous union by aiming at normal occlusion, I let good occlusion go rather than allow the patient to have a fibrous union. I have a case in mind in which the ideal was aimed at, and after six months there is still fibrous union. If in this case one had been content to let the mandible contract $\frac{1}{2}$ in. there would have been osseous union with a good masticating area, and the man would have been efficient in four months.

We must also take into consideration the value of the teeth which remain in the mandible. If the teeth are not of value, you can afford to contract the mandible, because a slight reduction in the mandible is of

advantage when making a denture. So when there is a slight loss of tissue I am more inclined to allow osseous union to take place, with a slight reduction in the contour of the mouth. At the back of the mouth I get over the difficulty by releasing the posterior fragment and allowing it to move forward.

The treatment of the scars also forms a large section in our treatment of these cases. We cut away the scar tissue freely from the bone by means of scissors, and adopt forcible stretching of the soft tissues by means of a plug of vulcanite. It is then remarkable the way in which these depressed scars will flatten out, and I am sure that by this means we shall avoid some plastic operations. Further, it is obvious that if the scar tissue is soft, a plastic operation, if necessary, will be more likely to be a success than if it is attempted with scar tissue adherent.

Another type of case is the "stiff jaw," which so often follows fractures of the ascending ramus. If you treat these in the ordinary way with the screw gag you exaggerate the deformity which they have. When they open their mouth it is with an inclination towards the affected side. We have got a splint for these cases which was designed by Captain Holt. There is a plate covering the upper and lower teeth, and by means of two screws we can bring about a parallel opening. It is put in and kept in by night and left out during the day. The way to treat these cases in which scars have to be stretched is to use continuous stretching over a definite period. You get a better result by stretching for twelve hours and then giving twelve hours' rest than by stretching for, say, ten minutes every three or four hours.

Another point I wanted to speak on was that of bone-grafting. Up to the present we have had two patients bone-grafted, and they are both going on successfully. I am extremely interested in this, as is also Sir Frederic Eve, for in the method of treating fractured jaws with loss of bone success will largely turn on the results of bone-grafting. If one can rely on the success of bone-grafts, clearly we shall be more inclined to aim at an ideal jaw.

Those who have been to the Croydon War Hospital will agree that there is nothing special about the treatment there. The cases of fractured jaw are treated on the same lines as fractures of other bones. The first necessity is cleanliness, the second is rest.

By the consent of the Colonel commanding the division, I have been allowed to bring for your inspection twenty of the patients, and I shall be pleased to explain to you afterwards the methods of treatment.

Under the rules of the Royal Army Medical Corps, we publish our papers in the *Journal of the Royal Army Medical Corps*, and it may interest you to know that the first account of the Croydon War Hospital cases will probably be published in the April number. The hospital is to have 160 beds, and at present I have working under me two doubly qualified dental lieutenants; and as our patients become convalescent they are to be moved to another block, so there is no reason why at any time we should not be dealing with 250 patients. Everything has been done there that could be done for one's comfort. Sir Frederic Eve, the Consulting Surgeon to the Eastern Counties, takes a keen interest in the work.

I should also like to thank General Russell, who is present, for the great help he has given me. He made an inspection of the Hospital the other day, and personally examined every one of the cases.

SIR FREDERIC EVE.¹

In visiting various parts of the Eastern Command my attention has during the past fifteen months been constantly drawn to various problems connected with the treatment of gunshot fractures of the jaws. The assistance of the surgeon is only exceptionally required if treatment by a dental surgeon has been carried out skilfully from the outset of the case. Otherwise he may be called upon to divide the mandible for osseous union in a bad position. Not infrequently the union is by dense fibrous tissue; this can usually be divided by scissors, or by a blunt-pointed bistoury passed through a small opening in the original scar. In one case, where a considerable length of the mandible had been lost and firm osseous union had occurred, I divided the mandible by the Z-shaped method, and wired the fragments together after separating them to the desired amount. This gives a firm bony union where a gap would otherwise be left. It is, however, essential that a splint should be provided beforehand, so that the wiring of the fragments may be made in such a way as to bring about correct approximation of the teeth. Very severe cases occasionally come under observation where the whole of one side (except part of the ascending ramus) or a lesser amount of the mandible has been shot away, with much destruction of soft parts. The opposite side falls inwards and

¹ Lieutenant-Colonel (Temp.) R.A.M.C., Consulting Surgeon, Eastern Command.

downwards. Deformity may in these cases be prevented and the patient's condition made more comfortable by placing stays of some rigid wire, such as phosphor bronze, across the gap. The edges of the wound in the soft parts may perhaps be excised and partly closed. The method is illustrated by the accompanying illustration¹ (fig. 1) from a case in which I removed one half the mandible, with the exception of the upper part of the ascending ramus, for epithelioma. It may be noticed that the proximal ends of the wires were firmly driven straight



FIG. 1.

Radiogram from a case in which the left half of the mandible, with the exception of the upper third of the ascending ramus, was removed for epithelioma. The right half of the mandible was kept in position by two stays of phosphor bronze wire; the proximal ends of the wire were driven into the left ascending ramus and their distal ends were passed through the symphysis and twisted. This arrangement permitted of the upper wire being removed about three weeks after the operation. The lower became embedded in fibrous tissue. The patient was shown, nine weeks after the operation, at the Clinical Section of the Royal Society of Medicine. He could open and close the mouth freely, and could masticate soft food on the right side.

¹ From the *Proc. Roy. Soc. of Med.*, 1914, viii (Clin. Sect.), p. 14.

into the bone, the distal ends being twisted. This allowed of the upper wire being removed, while the lower was left and became embedded in scar tissue. Patients can open and close the mouth readily from the first and displacement is prevented.

I devised this method many years ago for a case of resection of a central fibro-sarcoma, and left in two silver wires. These became completely embedded in fibrous tissue and had given rise to no inconvenience when I saw the patient with Dr. E. J. Reynolds, of East Dulwich, some twelve years after the operation.

I adopted this method in the case of a Belgian officer whom I had to treat under conditions in which I could not obtain skilled dental assistance for the making of splints. The median part of his mandible and the lower lip had been blown off, and the two halves of the mandible had fallen together, forming a "parrot-jaw"; the mouth was circular, about the size of a florin, and could not be closed. I divided the mandible in the middle line, put in wire stays to keep the fragments apart, and restored the chin and lower lip by a flap from the neck. The result as regards the lower jaw was quite satisfactory, the wires becoming soundly embedded in fibrous tissue and the chin being restored. The new lip, although at first it could be approximated to the upper, subsequently became somewhat drawn down by contraction of the dense scar tissue in the neighbourhood. This will usually occur, and must be guarded against by taking, after allowing for contraction of the graft, much more tissue to fill up gaps than may appear necessary. These methods, although by no means ideal, may profitably be borne in mind by the military surgeon, who often has to employ such means as he finds for the moment obtainable.

The ideal method for bridging over gaps in the mandible is, of course, the transplantation of bone, but this requires special conditions. The wound must have soundly healed and the soft parts must be sufficiently abundant and dense to allow of their being peeled off the injured bone without the mouth being opened. If the teeth are not correctly approximated the fibrous union should be stretched by preliminary splinting or by an operation, for I have found that if this is attempted at the transplantation operation the mouth will be opened in the manipulations necessary in widening the gap between the fragments. Great care is necessary in refreshing the ends of the bone on each side of the gap. The whole operation is rendered much easier if a splint be firmly cemented to the teeth across the gap. The surgeon

need not then trouble about the position of the teeth, the fragments being firmly held can easily be refreshed, and the length of the necessary graft can be measured accurately with calipers. This method has been adopted by Mr. J. F. Colyer and myself in two cases we have treated at the "Jaw Section" of the Croydon War Hospital. In both instances I transplanted portions of the eleventh rib. In one, the edges of the sections of the lower jaw were quite vertical, and the piece of rib fitted so accurately that it was only necessary to fix it to the mandible



FIG. 2.

Radiogram showing an ununited fracture with considerable loss of bone in the left horizontal ramus of the mandible. The teeth are occluded by a metal splint, and a portion of rib has been grafted into the gap in the bone. This is retained in position by two wire nails.

by two wire tacks, as recommended by Murphy (fig. 2). In the second case one free end of the mandible could not be cut square without considerable sacrifice of bone; therefore the rib and mandible were perforated respectively on each side of the bridge and fixed together with silver wire loops. In both cases the wounds healed normally.

The periosteum was stripped off the rib, as it has been shown that it restricts the osteoplastic process.

I have not yet seen a case in which plating of a gun-shot fracture of the mandible seemed advisable. We had at Croydon, however, a patient with a fracture of each horizontal ramus which had been treated by plating elsewhere. The fracture had firmly united, but in bad position, and sinuses led down to the plates. These were removed and the site of union was divided on each side with a fine saw to allow of correct approximation of the teeth by splinting. I can hardly imagine a condition in which the application of a plate is to be preferred to that of a splint or wiring, except as a makeshift.

In conclusion, it may be pointed out that in the earlier stage of treatment of severe comminuted fractures of the mandible special care should be taken to preserve fragments of bone which may be viable. If they necrose, removal may be effected later, and meanwhile they may prevent displacement while new bone is being thrown out by the periosteum.

Odontological Section.

President—Mr. H. BALDWIN.

(March 6, 1916.)

ADJOURNED DISCUSSION ON WAR INJURIES OF THE JAW AND FACE.

Mr. WILLIAM HERN.

SINCE the early days of the War I have been engaged in assisting my surgical colleagues at No. 3 General Hospital, Wandsworth, in the treatment of war injuries of the jaws. We have had quite a considerable number of cases, some of them severe and difficult, some easy and straightforward. Shortly after I commenced my work there my colleague, Mr. Hubert Daw, came to assist me. As there were such a large number of Territorials waiting for plates to make them efficient for service we began providing them with ordinary dentures, but we soon had more of this work than we could cope with. However, matters went better when Mr. West joined us and took over the provision of ordinary dentures under the War Office scheme. Mr. Daw and Mr. West have not only helped me with the ordinary dental needs of the patients in the hospital, but have had a hand in many of the cases of which I propose to speak.

You, Sir, the Opener of this discussion, and other speakers, have sufficiently indicated the special characters of these war injuries as compared with those in civil practice by alluding to the comminuting effect of missiles on the bones of the jaws, to the frequent loss of substance, especially at the point of exit of the bullet, to the frequency of multiple fractures, to the considerable displacements which occur, to the liability of foreign bodies, such as fragments of bone, teeth, or missiles, being present between the fragments, and to the serious lacérations of the soft tissues of the mouth and face. One point, however,

I may be permitted to emphasize—namely, that from the nature of the injuries to bone and soft tissues there results, in the majority of these cases, a very septic condition of the wound, and a septic discharge is poured into the mouth for a considerable time during the early stages of the healing process. In some, necrotic sequestra of bone are exfoliated for a considerable time. Obviously, therefore, these war injuries cannot be treated in the same manner as cases occurring in civil practice.

The treatment must be divided into two main stages: (1) The correction of displacements and support of the fragments whilst the healing of soft tissues and bony union is taking place; (2) the fitting of some prosthetic apparatus for the replacement of the lost parts, the restoration of the function of mastication—possibly also of speech—and the reduction of disfigurements. The interval between these stages—sometimes a considerable one—should be devoted by the patient to the stretching of scar tissue and massage of any cicatricial bands which may have formed during the healing of soft tissue. I have been astonished at the degree of suppleness of cicatricial tissue which such treatment can produce if persevered in over some period of time.

On account of the character of the wounds, it is important, I think, that the splints and other apparatus used in the first stage of treatment should be simple and aseptic in design, and be applied in a way which will avoid the damming up of discharges. The mandible should not be fixed to the maxilla if that can be avoided, and the apparatus should cover the minimum possible of gum surface. The occlusal surfaces of the teeth should be free. These requirements seem best met by some sort of wire splint. A Hammond, or a modification, an Angle arch attached to bands on back teeth, are all cleanly and effective where firm teeth exist on both sides of the fracture; where these conditions do not exist the cradle wire splint suggested by Mr. Lewin Payne, which is virtually a double Hammond, is very useful, and in some cases the trough or a capping splint might have to be resorted to.

In view of the seriousness of the injuries, I have been surprised at the small number of cases which cannot be treated in the early stages by some simple wire splint.

The cases of fracture of the maxilla which have come under my notice have been caused by bullet or shrapnel; but I have not yet encountered cases of such extreme severity as our French colleagues showed in some of their photographs. Certain portions of the alveolar border of the teeth have been shot away, with or without perforation of the palatal portion of the bone. Some disfigurements have been caused

by laceration of soft tissues and loss of bone, but they have caused no greater difficulty in treatment than the construction of a plate or obdurator, or both, to close the palate wound. I accept Mr. Lewin Payne's six types of fracture of the mandible as fairly covering these injuries from a clinical point of view.

I have seen several cases of serious injury to the mandible, and I should like to refer to some varieties which I think present the greatest difficulties in treatment: (1) Single or double fractures at the angle, or behind the last molar tooth; (2) bilateral fracture of the mandible in the mid-horizontal region; these cases present themselves with the horizontal ramus ploughed through on both sides, with considerable loss of bone substance and of teeth, and, perhaps, an additional fracture at the symphysis, owing to the impact of the missile on the horizontal ramus; (3) fracture involving the loss of the front part of the mandible, including the six or eight front teeth; (4) fracture involving the loss of the whole of one side of the horizontal ramus and perhaps part of the ascending ramus.

In single or double fractures of the angle, an exceedingly difficult condition may arise from the position and line of fracture in relation to the attachment of the jaw muscles. The posterior fragment is displaced forward and upward, and the anterior fragment downwards and backwards. In these cases, as there is nothing to hold on to in the posterior fragment, a capping splint extended backwards to hold the ascending ramus in position will be necessary, and in some cases resort must be had to the fixation of the lower jaw to the upper.

I have met with several cases of bilateral fracture of the horizontal region of the mandible. In these there is usually a great loss of bone substance, the loss being usually greater at the point of exit than at that of entrance of the missile, and there is often much comminution of bone. In connexion with these shattered cases, I consider it very important to retain every piece of living bone. I remember one case particularly in which there was a ploughing wound from left to right, leaving a few incisors in front; these were attached to small fragments of bone, which were so freely movable that my colleague thought removal would have to be carried out, but as there was a fair vascular supply to the gums I advised that the teeth should be kept. The result proved the conservative treatment to be right, because there is a solid filling up with bone over a gap of $\frac{3}{4}$ in. on one side, and at the point of exit of the bullet on the other side, where the gap was larger, there is very good fibrous union.

Where fractures involve the whole anterior arc or front of the mandible, carrying away six or eight front teeth, including the chin, there is a very characteristic displacement—namely, the two fragments always fall to the mid-line and tend to meet in front, causing what has been termed a "parrot mouth." To prevent this, it is wise to secure a model as soon after the infliction of the injury as possible, and to make a splint to hold the two sides apart; I have used a Hammond splint for this purpose. Sometimes I have secured models before my colleagues have dealt with the soft tissues. It is important, I think, to prevent the inward falling of the teeth and the loss of occlusion.

I agree with Mr. Colyer that the great point is to get bony union if we can do so without serious loss of occlusion; but most of these cases have had so much shot away that it means a great degree of obstruction to the tongue, and absolute loss of occlusion of upper and lower molars, to allow the two sides to fall together and join up with bone. It is therefore, in my view, wiser to prevent the falling inward of molars, to preserve their occlusion with the upper, and afterwards to make an artificial restoration of the lost teeth and bone.

In regard to the question of whether a movable or a fixed prosthetic apparatus is best for the patient, I regard the movable one as preferable if it can be made secure and firm enough for mastication. The life of the remaining natural teeth, on which the stability of the apparatus and the comfort of the patient depend, will be longer; and the condition of the gums will be better with a removable than with a fixed apparatus. One disadvantage of the military hospital is that, owing to military exigencies, patients must sometimes be cleared out of hospital before their treatment is really complete, and in this way I have lost sight of unfinished cases.

With regard to the policy of extracting teeth on each side of the fracture, which Mr. Colyer advocates as a routine procedure in all his cases, I think it may be advisable, and even necessary, to extract a tooth in the line of fracture, but I fail to see why we should do more than that in every case we meet. I can show you a case of triple fracture of the mandible in which six teeth would have been unnecessarily removed from one patient by such procedure. I regard the loss of the mutual support of contiguous teeth in the fracture line as a disadvantage, quite apart from the advantage of preserving the teeth for masticating purposes. Possibly in cases of ununited fracture, the taking out of teeth near the line of fracture might set up a bone-forming process for the

repair of the socket, and might thus to a small extent expedite bony union. In the cases of fractures seen early, I regard that measure as unnecessary.

As these war wounds are so septic, I agree with Mr. Lewin Payne that efficient drainage is a *sine qua non*; also thorough irrigation of the wound at short intervals. Peroxide of hydrogen, 5 to 10 vols., and common salt solution are of great service for this. Equally important is a regular shampoo of the gums with a tooth-brush; this prevents gingival troubles and oral sepsis. I have known patients who did this retain quite healthy gums throughout the treatment, notwithstanding several compound fractures of the mandible.

I have seen many cases of what Mr. Colyer calls "stiff jaw," which he considers to be due to fracture of the ascending ramus. The last case of the kind I saw was shot through from left to right, 1 in. in front of the auditory meatus, and probably part of the coronoid process was injured by the bullet. But there are cases without fracture of the ascending ramus, in which the bullet has perhaps grazed the bone, and ploughed up the muscular tissue of the masseter or internal pterygoid muscles, which have been followed by this stiffness. I have usually treated them by intermittent spring force, using the spring clip which I show you.

My experience of bone-grafting has been very slight; I have suggested to my colleagues some cases for treatment, and I think I have one now pending. I have seen one returned prisoner from Germany, the front of whose mandible had been shot away. The German surgeon did his best for him whilst a prisoner by taking a piece of his tibia and bone-grafting it, but the operation, unfortunately, failed.

I should like to join in a hearty meed of thanks which has already been expressed to our French colleagues for the care they have bestowed upon the cases shown in the Museum, and the help they have given us and to our discussion by their presence.

We had one curious foreign body case, photographs of which I will show you, kindly lent for the occasion by my colleague, Mr. Kellock, under whose care the patient was, in which a man was hit by two pieces of shrapnel in the two jaws. A skiagram showed what apparently was a round piece of shrapnel. On operating, it was found to be a tunic button in the right antrum. The man was not wearing his tunic when he was struck, but it was lying on the parapet of the trench, and the shell burst just in front whilst the man was resting. The missile had evidently taken one of the buttons off the tunic and propelled it into his face.

Lastly I wish to endorse Mr. Lewin Payne's four points, which he mentioned at the end of his opening address, as I consider they are very important.

MR. MONTAGU HOPSON.

I did not anticipate that I should be called upon so early in this resumed discussion. Adverting to what Mr. Colyer said at the meeting last week, I think that, although some of us will not accept without some reserve all those points which he postulated as being essential in the treatment of these cases, we must all admit that the results which he has achieved by adopting the methods he advocated, as evidenced by the cases he showed us afterwards, lead to success in his hands. What was novel to me was the way in which he dealt with some of the cases of fracture of mandible in the molar and premolar regions with loss of substance. He removed the upper teeth and allowed the posterior fragment to swing forward. And he said he was prepared to sacrifice some degree of occlusion so as to secure bony union. I admit I am with him when he does that—i.e., when there is but slight mal-occlusion; but surely the degree of mal-occlusion so achieved must be taken into consideration. If that is likely to be great I prefer fibrous union, and it is wonderful how dense this sometimes is. In one case in which I co-operated with my colleague at Guy's Hospital, Mr. Hughes, we had that difficulty. The patient had been under treatment at a certain base hospital, and had been discharged to his *depôt* for duty with an ununited fracture of the mandible in the region of the first molar on the right; the larger fragment on the left was badly displaced to the right, overlapping the smaller fragment on its inner side. Of course, the patient could not masticate the food as supplied at the *depôt*; indeed, he had not sufficient power to prevent the withdrawal of my finger when placed between his teeth. The treatment adopted was as follows: Caps were struck to cover his upper teeth on the left, and others for his lower teeth on the right, and by means of intermaxillary traction with rubber bands, from the upper on the left to the lower on the right, the larger fragment was brought across into its correct position. There was one feature in which I came to grief, and it should be mentioned. The caps were made of German silver, and I inserted them in the mouth without having had them gilded. The reaction, however, between the oral secretions and the base metals was so active that I was compelled to remove them and substitute others. When the

large fragment had been brought into place it was found that there was a gap of about $\frac{3}{8}$ in. between the two fragments. It was a question whether we should sacrifice a certain amount of occlusion, and secure bony union by bringing the two ends together, or be content with fibrous union. We decided to sacrifice a little occlusion. A factor which influenced me in coming to this decision was this, and I think it should always be taken into consideration under such circumstances: the patient needed a lower denture owing to the loss of teeth, and I saw my way to affording him a good masticatory apparatus afterwards by means of a denture. Mr. Hughes wired the fragments by an external operation; three weeks later there was excellent function, and the man returned to duty.

I have had experience of bone-grafting in only one case, that of an officer, who has very kindly come here to-night for your inspection. We saw him first two months after he had been wounded. There was a fracture at the angle on the right side, which had already united in not a very good position, producing a slight open bite. His major jaw injury was on the left side; there he had lost a segment of the mandible containing six teeth | 1 2 3 4 5 6. There was a large open granulating wound of the left cheek and neck, besides other injuries. I made a gold cap splint, covering the teeth in the larger fragment on the right, and the two remaining molars in the smaller fragment on the left. The two series of caps were united by a large Badcock screw, by means of which the fragments were brought into approximate occlusion with the upper teeth. A tendency for the smaller fragment to be pushed backwards and outwards by the thrust of the screw was overcome by intermaxillary traction. When the wounds of the soft parts had completely healed, Sir Alfred Fripp grafted $2\frac{1}{2}$ in. of the patient's sixth rib into the gap. Each exposed end of the fragments was notched to receive the pointed ends of the portion of the rib. It was neither wired nor pegged. The graft was made, of course, from the outside, great care being taken not to open into the cavity of the mouth. It is nearly four months since the operation, and the radiograms show good bony union. The splint is still in place, and I think it wise to leave it there for some time yet. There was one point in connexion with this case which has some bearing on that so strongly advocated by Mr. Colyer—viz., the removal of the teeth adjoining the line of fracture. Here I had only two teeth in the smaller fragment, the second and third molars, both invaluable for securing a good hold. After my splint had been on for some weeks a portion of the anterior wall of the socket of the second molar

exfoliated, exposing the root of the tooth, and it is true that bony union at this distal end of the graft has been much slower than at the other end, and it is possible that that tooth may ultimately be lost.

In a purely military hospital the efforts of the staff are mainly directed, and no doubt quite rightly so, towards making a man an efficient fighting unit in the shortest possible time. There are many cases, however, in which the injuries are so severe that the victims will never return to the fighting line again. Such have to be evacuated from hospital as soon as possible to make room for more useful military material; they often receive an early discharge from the Army, and drift to us in civil hospitals and private practice to see what can be done for them. As the President mentioned, some more adequate provision must be made for these men than exists at the present time. I have under my care at the moment a man who has been discharged from the Army. He has lost practically the whole of the body of his mandible. There remains but a small portion on the left side containing his second molar tooth. He cannot chew anything. The whole of his right cheek and the floor of his mouth consist of dense scar tissue. By means of inclined planes I succeeded in releasing the small fragment on the left, so that I can now push it outwards into occlusion with his upper teeth. I then made him an appliance which consisted of a lower plate with a few teeth, engaging planes on the left side, and a single spring on the right attached to an upper plate. I found, however, that when the planes engaged on the left side, the spring exerted so much traction on the scar tissue of the opposite side, that no spring was strong enough to keep the lower plate down. For a month now he has been wearing a vulcanite Gunning splint; this enables me to make a *point d'appui* of his maxilla, and by adding layer after layer of base-plate gutta-percha to the outer side of the splint I have already succeeded in stretching the cicatricial tissue to a marked extent. It is now proposed to divide the more obstinate of the fibrous bands, and I hope to insert an appliance working with a single spring, such as I essayed in the earlier stages.

I have one other interesting case, not unlike one Mr. Hern showed us, that of an officer who was injured at the beginning of December. He is under the care of Mr. L. A. Dunn. When he came all he had remaining on the left side was a portion of the ascending ramus and on the right a small fragment containing the third molar tooth. It is the kind of case concerning which Sir Frederic Eve last week uttered a warning. Unfortunately, every fragment of bone had been removed in France, before he was sent to us; no dental surgeon had a chance to

see what he could do. When he arrived he had an incision 4 in. long in his neck, and there was a cicatrix rapidly closing in and dragging on the soft tissues. His chin had gone. He was so ill that it was a long time before we could do anything. After a time I made an appliance such as Mr. Hern mentioned. I inserted a skeleton lower plate attached by springs to an upper with a downward and forward thrust, and I have added layer after layer of gutta-percha until I have got it an inch thick, and it is now necessary to make a new plate. We are getting the chin well forward. He can now speak, swallow, and deal with his saliva, none of which he could do before.

It is obvious that these injuries cannot be treated by the surgeon alone, nor by the dental surgeon alone; complete collaboration is needed from the very commencement, and I cannot conclude without expressing my profound regret that the Army Medical Department has not even yet made adequate provision for their treatment, despite warnings which were given and offers of assistance which were made, at the very commencement of the War.

MR. STANLEY MUMMERY.

I will confine the few remarks that I have to make entirely to one point in the treatment of jaw injuries. I refer to the methods of dealing with missing portions of the mandible, where a larger or smaller part of the arch has been destroyed, leaving a gap in its continuity.

From the point of view of treatment, I think these cases fall naturally into two categories: (1) Those cases where the missing portion does not exceed from $\frac{1}{2}$ in. to $\frac{3}{4}$ in. in breadth; (2) those cases where it does exceed this amount, and may even involve half or more of the mandible.

In the latter class of cases, when large portions of the bone are missing, it is obvious that artificial restoration is the only course possible in the majority of instances. In favourable cases, where sound bone exists on either side of the gap, bone-grafting promises to give most satisfactory results, and it is to be hoped that when more experience of this operation has been gained, bone-grafting will take the place of artificial restoration in a large majority of cases.

It is, however, in the treatment of those cases coming under the first category, where the loss of bone does not exceed $\frac{3}{4}$ in., that there is most room for discussion. The only known method of dealing with such injuries, up to a few years ago, was to bring the fractured ends together

and allow them to unite, wiring them together if necessary. This method had the one advantage of producing a sound jaw, but from the point of view of efficiency it was extremely bad. The bite of one or both sides was completely destroyed, and the teeth rendered practically functionless.

Mr. J. F. Colyer has introduced an original method of overcoming this difficulty by allowing the ramus on the injured side to swing forward to restore the deficiency, and so maintain the breadth of the arch. The results Mr. Colyer obtains by this method are extremely satisfactory in comparison with the older method, as was amply demonstrated by the cases he showed here last week. The only disadvantages of Mr. Colyer's method appear to me, firstly, the sacrifice of teeth on that side to allow of the swinging forward of the ramus, thus rendering that side practically functionless in the molar region, and secondly, the failure of this method for cases with loss in the median line near the symphysis.

The alternative method is that adopted by many workers in France, and some in this country. It consists in the separation of the fragments and their fixation in normal occlusion with the teeth of the opposing jaw, in the belief that osseous union will take place across the gap. Under favourable conditions I believe that osseous union will take place in the great majority of cases. These favourable conditions are: absence of sepsis and gradual separation of the fragments. By gradual separation, I mean that the parts should not be forcibly separated to the full extent ultimately desired at one operation. Small particles of bone with some periosteal attachment are invariably left scattered in the tissue between the fractured ends, and such particles become the centres for bony deposit. Where the parts are forcibly separated a definite rupture of the tissues occurs, whereas when the separation is gradual, the parts have time to stretch, and the new osteoblastic tissue is drawn across the gap. This may sound like pure conjecture, but clinical evidence bears it out. Those of you who have seen Dr. Hotz's work in Paris will have noticed how successfully he employs this method, and what satisfactory results he obtains. There is one point, I must own, in which this method compares unfavourably with Mr. Colyer's; it is that of time. It obviously requires a longer period to obtain firm bony union across a gap than it does when the ends of the fragments are in contact. Upon this question of time I know Mr. Colyer lays great stress, and from the point of view of the Army it is perhaps an important one. From the patient's standpoint, however, I do not think there can be much doubt that the separation method produces the best results.

Mr. G. NORTHCROFT.

You, Sir, have expressed the opinion through Mr. Payne that a general review of the whole subject is not sought for from every speaker, but rather a relation of his own experiences, the correlating of which with others may help to elucidate many of the difficult surgical and mechanical problems that await solution.

As far as classification goes, two great groups or types are surely seen in fractures, "*with and without loss of substance.*" These may both be divided into simple or multiple; and these again under the various displacements, either singly or in combination. In my experience displacement that is only lateral or vertical rarely occurs, but rather in combination with inward or outward, upward or downward, tilts. Cases without loss of substance present no grave difficulties if taken in time.

As far as treatment where loss of substance is concerned, I think these cases are dividing themselves very much according to the way in which they can be dealt with. If the loss occurs between remaining sound teeth in both fragments, immediate attention to prevent displacement by cicatrization should be undertaken and a bone-graft attempted later, or fibrous union allowed to occur of such a nature as to bear a denture, so that the jaw is quite steady when a denture is in place. The more difficult case to deal with is when there is no tooth remaining in one, say the posterior, fragment, so that in attempting bone-grafting failure is rather invited by the impossibility of immobilizing the posterior fragment. Should plating be attempted in such cases? If the loss is not too great, the angle may come forward, and, although swinging somewhat inwards and upwards, may become united, the other side of the jaw having been retained in correct occlusion. If the loss of substance is too great, the remaining fragment must be immobilized in occlusion, or intermaxillary traction applied until cicatrization is complete, the latter in such a manner that only horizontal strain occurs when the mouth is at rest. The question that has to be answered by experience is whether it is better to immobilize a man's jaw with possible intervals for, say, six months, or whether intermaxillary traction will be powerful enough to overcome contraction in all cases. I wish heartily to endorse all that Dr. Hayes said about plastic operations being performed without consultation with the dental surgeon; a man under my care had a typical parrot jaw, but he had lost much soft tissue, and I imagine the remaining edges had

been sewn together, so that if the mandible had been expanded, the patient would have had a permanent open mouth, never being able to close his lips. This difficulty was overcome by extracting the lower teeth and inserting a denture to occlude with the upper teeth.

I present the photograph of one patient who had his right maxilla fractured, and the mandible in four places, with loss of substance on the right and no remaining tooth in the posterior fragment; the occlusion is here being maintained by intermaxillary traction until such time as flanged dentures can be inserted with a reasonable prospect of success.

Mr. Colyer has referred to making these men speedily efficient. Of course, one's idea of speed may differ, but many of these cases are necessarily very tedious, and the injuries have been so severe that future military efficiency is out of the question.

I have also designed a temporary cap splint which is uncemented, has the front cut away, which is easily removable (often an important point), being ligatured to three or four teeth round loops on the lingual side and round vertical wires on the buccal side. It can be used as a single splint for reducing both vertical and lateral displacements, and enables one to see that they *are* reduced, or may be used in connexion with an upper cradle if it becomes necessary to immobilize the jaws. As a practical point in the construction of cap splints, I might say that they should always be made in two halves, reinforced and soldered.

The lip-distending frame illustrated on the screen is found very useful. Where photographs are required for oral display, this frame does away with the marring of them by fingers or cheek retractors. It is made in three sizes, and may be put into position by the patient himself.

MR. NORMAN BENNETT.

It has been a common practice to divide fractures into those where there is bone missing and those where there is none missing. For practical purposes, in regard to war fractures, we are only concerned with those in which bone is missing. A very practical classification, I think, consists in cases from France and cases from the East. The cases from France reach us now tolerably soon, and exhibit the conditions as they arise soon after the injury. Cases which reach us from the East come after three months of what we may describe as "glorified" first-aid treatment, i.e., their septic conditions have been dealt with,

and also their surface and skin wounds—perhaps too much dealt with; but their bony lesions have been practically disregarded, except as regards the removal of necrosed portions. The difficulty of treating them afterwards is, of course, very much enhanced.

The discussion has mainly centred round bony union versus fibrous union. The ideal is bony union in good occlusion; and the question is, what departure from the ideal is permissible, and in what direction? Mr. Colyer considers that the chief thing to seek is bony union, even if there result some lack of normal occlusion. My experience does not lead me to subscribe to that view. If there is any considerable loss of substance, we can hardly hope for bony union at all; if, however, there has been only a small loss of substance, so that only a small error of occlusion will arise, then I think we may still hope for bony union. In that respect Mr. Stanley Mummery hit the right nail on the head when he said that gradual methods are likely to yield the best results. If two pieces of bone are separated by a considerable interval, it is a good deal to expect that the interval will be bridged; but if you allow callus to form and to consolidate, and then proceed to separate the parts by stretching, it is logical to expect permanent bony union. I speak of cases which are only half finished, and that is all that can be done by most of us; it is only the simpler and earlier cases that are complete.

As regards apparatus, I have a predilection in favour of removable rather than fixed apparatus. Mr. Dolamore is very successful with fixed cemented cap splints, but I think the latter are only desirable under two opposite conditions: (1) That in which there is a large portion of bone missing, in which you fix the parts to get fibrous union; (2) the condition in which there is only a very small portion missing, in which you also hold the parts and will certainly get bony union. But to apply a cap splint under an anæsthetic to a patient who is not sufficiently anæsthetized for muscular relaxation, whose mouth is full of saliva, I find very difficult, as you are trying to open the mouth with a gag, to keep the teeth dry, and mix up your cement before dryness has disappeared. I have achieved better results by more gradual methods.

There is another point to which I want to refer before showing three cases which I still have in hand—namely, the position of the fragments and the classification which Mr. Lewin Payne has suggested. It has been impressed upon most of us that the fractures of the mandible sort themselves out into three classes as regards position: (1) Fractures in the premolar region; (2) fractures of the angle and

ascending ramus; (3) fractures in which the front part of the mandible is lost between the canine regions. It is puzzling why nearly all the cases exactly conform to one or other of these groups. Still, it seems fairly obvious. If a projectile passes in more or less horizontally from before backwards it kills the man; if it passes transversely through both angles it probably kills him. If it passes obliquely, it produces a very common condition of fracture in the premolar region on one side and the angle on the other. But if it passes transversely, but so far forward as not to kill, it removes the front part of the man's mandible. With regard to the direction taken by the bullet, I should like to mention one peculiar case. The man was running in a charge, and the bullet entered his open mouth. It fractured the alveolus of the right maxilla, passed the inner surface of the ascending ramus of the mandible, and came out behind his mastoid process, making a long track, but doing comparatively little harm; the patient made a good recovery, except for some facial paralysis.

MR. PERCIVAL P. COLE.

I shall take as the text of my remarks some statements and expressions of opinion voiced by previous speakers, bearing upon treatment and the personnel responsible for such.

Discriminating conservatism as regards the teeth is an early and important factor in the treatment of these cases. As Dr. Hayes has so succinctly put it, "the presence or absence of a single tooth on a fractured portion may suffice to change entirely the method of treatment"; and I may add that the conservation of one or two particular teeth will render relatively easy what would otherwise be a task beset with difficulties. So convinced am I of this that I have had root treatment carried out on the operating table. It is a question bearing equally on restoration of function after operation as after injury, and my views on this question and those of my colleague, Mr. Bubbs, have been more fully set forth in a recent paper.¹ Some members of this Section may be inclined to attach greater value to the teeth than does Mr. Colyer, and so to deprecate the adoption of a policy of extraction, however consistent that may be, for the treatment of fractures. I refer to that type of fracture in which the upper molars are extracted to free the posterior fragment as a fundamental principle of treatment, and to secure rest to those ununited fractures of, as I gather, the same type

¹ *Brit. Med. Journ.*, February 19, 1916, p. 268.

which are admitted with a see-saw movement. If the upper teeth are capable of depressing the posterior fragment, it should surely be possible to maintain that fragment in its correct and depressed condition without resort to extraction in the opposing jaw. I have under my care at the present moment two fractures situated in the position indicated by Mr. Colyer. In the one case the posterior fragment contains two teeth; in the other case the posterior fragment is edentulous. In both cases the posterior fragment has been efficiently controlled in its correct position, in the former case by means of a fixed interdental splint cast in aluminium, and in the latter case by means of a cast aluminium Gunning splint with an adjustable flange lined with soft rubber.

A consideration of the combined contributions of Sir Frederic Eve and Mr. Colyer, together with Mr. Colyer's frank renunciation of ideals, would seem to indicate that the lines of treatment adopted have been dictated by military necessities, and with the object of reducing surgical intervention to a minimum—that, in effect, a short cut has been taken to the attainment of passable, but confessedly imperfect, results. However such a gospel may be justified by military exigencies, it is a gospel the broadcast promulgation of which will not assuredly subserve the cause of real progress.

Mr. Colyer's attitude as to the question of occlusion *versus* union seems to have been, and apparently still is, regulated by his scepticism as to the efficacy of bone-grafting. We are informed that 200 cases have been through Mr. Colyer's hands, and one cannot help thinking that, had Mr. Colyer been a general surgeon, his scepticism would long ere now have been dissipated or confirmed. A case under my care has an important bearing on this question. The patient had been treated by a surgeon and a dental surgeon whose views must have exactly tallied with those held by Mr. Colyer. He certainly had bony union, but his occlusion was hopelessly at fault. He—reprehensibly enough, no doubt—refused to share the complacent views as to his condition held by his surgical and dental advisers, and was sent to me as a last resort. So intense was his dissatisfaction that he pleaded with me to undertake any operation, however risky and however doubtful of success, that would help to remedy his functional incapacity and his deformity. I unwillingly acceded to his request, and undertook an extensive operation which, I am pleased to say, shows every prospect of success. I emphatically dissent from the views held by Mr. Colyer on this question of occlusion *versus* union, and my views have been more fully set forth, together with those of my colleague, Mr. Bubbs, in the paper previously mentioned.

Next, as to the important question of sepsis in its relation to the treatment of bony deformities of the jaws. I have personal knowledge that there is a widespread notion that reduction and retention of misplaced fragments should not be attempted in the presence of sepsis. Mr. Colyer's attitude is not easy to define in this matter, but he seems to favour a certain measure of delay, for he says: "We never think of fixing the splints until we are certain we have got rid of all sources of sepsis." I desire most emphatically to associate myself with the strong expressions of opinion on this question that have been so ably voiced by Mr. Lewin Payne and Dr. Hayes. Early reduction and retention of fragments in jaw injuries, far from being disadvantageous, are effectual means of combating sepsis, and add materially to the comfort and well-being of the patient. It is difficult to exaggerate the importance of this question, for misconceptions regarding it have been responsible for much suffering and much unnecessary treatment. It is a view widely held by the surgeons of the Royal Army Medical Corps, and I regret to say it is a view held by not a few dental surgeons, especially among the younger generation—among those, indeed, whose experience of mouth surgery is, one would think, scarcely sufficient to justify a dogmatic expression of opinion on such a point. I have been told by military surgeons of large experience that their dental colleagues have refused to intervene in the reduction and retention of bony deformities until sepsis has been thoroughly allayed. Upon this false foundation has been built a superstructure of error. To such an extent is this the case that by far the greater part of the sum total of attention and endeavour devoted to these war injuries has been centred not upon the treatment of fractures but upon the treatment of neglected fractures. This debate has provided ample confirmation of this statement, as have numerous drawings and appliances which have been exhibited during the past fortnight. Speaker after speaker has been an unwitting but damning witness to the truth of my contention. They have recited the tale of their exploits with intermaxillary traction, jackscrews, and I know not what. They seem to revel in the application of appliances that push and pull, elevate and depress. Their complacency is to me perfectly astonishing, and their bland acceptance of the existing state of affairs augurs ill for the introduction of any immediate and satisfactory measures of reform. Even Mr. Colyer does not regret that many of the patients with whom he has had to deal have been injured six months or even a year. The treatment of established deformities is undoubtedly of some importance as things

are, but far more important is it to prevent deformities from becoming established. I contend that the necessity for any apparatus which has for its object the reduction of deformity by applied force is, in the vast majority of cases, a frank confession of previously and avoidably defective treatment. This debate has been to me most disappointing in that little or no protest has been made against initial deficiencies in treatment which have such a profound influence on the future progress of these cases. I do not propose to detail my personal achievements, but shall endeavour to set forth my views as to the causes of these deficiencies—to follow up destructive by some measure of constructive criticism. Until quite recently these cases have been kept too long Abroad. There, owing to misapprehensions as to sepsis, the lack of skilled dental surgeons in the Army, and the ignorance of the general surgeons of the fundamental principles of treatment in these cases, their lesions have not received that special attention which would have laid the foundation of a successful issue. Stomatological centres should have been established in the various countries where our troops are fighting, or the cases should have been transferred immediately to Britain with the least interference possible. As regards France, at any rate, I have been given to understand that the latter plan will be adopted.

The next question that arises is as to the personnel responsible for treatment and the advantages to be derived by the segregation of these cases. It is readily granted that the ordinary fractures in civil practice are best treated by the dental surgeon. War injuries, however, are different, and the rules of civil practice afford no guide to the procedure in such cases. The lesions are frequently such that surgical intervention of varying extent is necessary at some stage. In the more severe cases, the casual surgery alone is such that no practising dental surgeon whose zeal did not outrun his discretion would care to undertake. The responsibility for methods of treatment that involve the question of life and death should, I submit, be vested in the operator, be he operating surgeon or operating stomatologist. In this country the operating dental surgeon does not exist, and the responsibility is usually, and I think rightly, shouldered by the surgeon. That this should have been freely and universally conceded would have appeared to me a foregone conclusion had not Mr. Colyer instanced as the first guiding principle of his treatment that "the lives of these patients have to be saved." I submit that in view of dangerous complications such as cellulitis of the neck and face, and the risk of secondary hæmorrhage, necessitating perhaps the tying of the external

carotid artery, that the best interest of the patient will be served by allowing the surgeon as heretofore to retain immediate control. For this reason, and because other wounds are frequently present, I am opposed to absolute segregation, but support the partial segregation of such cases in general hospitals, a course which, from personal experience, Dr. Hayes has found to offer the greatest advantages. A case bearing on the purely casual surgery that may be necessary in severe lesions is now under my care. The patient had had the greater part of both maxillæ shot away and was admitted with a suppurating wound in the neck with surrounding cellulitis. It was found necessary to enlarge the neck wound, and from it the whole of the ascending ramus was removed as a sequestrum. The body of the jaw was immovably fixed by cicatricial tissue which had to be freely severed in order to allow the mouth to be opened sufficiently for a reliable model to be taken. A fistulous communication between the mouth and neck wound was unavoidably produced, a condition which, however, experience has taught us is of relatively little moment.

In the treatment of these cases there lie at one end of the scale the purely surgical measures, such as bone-grafting and plastic operations, and at the other end the adaptation of a final prosthetic apparatus, which is a purely dental matter. Between these two extremes the treatment should rightly be shared by the general surgeon and the dental surgeon. It is here—in this, as it were, a kind of No Man's Land—that the frontiers of surgery and dental surgery abut, and the precise share to be taken by each is difficult to define. I would venture to suggest that what is required of the dental surgeon is:—

(1) That he be a skilful technician.

(2) That he be willing to make his services subserve the immediate, and anticipate the ultimate requirements of the surgeon.

Of the surgeon it should be required that:—

(1) He shall have a working knowledge as to the range and extent of the dental surgeon's possibilities.

(2) That he shall know and make known his requirements.

It is the lack of co-ordination in this stage of divided control that is, I am convinced, responsible for so many absolute and relative failures. The responsibility of the surgeon entails upon him definite obligations, the most important of which is that he shall call to his assistance at the earliest possible moment a reliable and skilful dental surgeon. It must be within the knowledge of most members of this Section that the services of the surgeon and the dental surgeon are only too frequently

consecutive and not concurrent. For this state of affairs the surgeon in immediate control of the case is directly responsible, and his course of action is such as to be an injustice to himself, the dental surgeon involved, and last but not least, to the patient. It is essential that no active step in treatment should be carried out by the surgeon until a definite plan has been formulated as a result of personal collaboration with the dental surgeon whom he chooses to requisition. Surgeons are, and have been, greatly to blame in their failure to appreciate what great assistance—nay more, what essential assistance—can be afforded by the early intervention of the skilled dentist, and it should have been one of the objects of this debate to bring this matter forcibly to the notice of the members of the Surgical Section. I am a surgeon myself, and it has apparently been left to me to emphasize and underline this failure on the part of the surgeons concerned, and I should like to make it clear that in my opinion this active collaboration is absolutely essential if success is to be attained.

My contribution to this debate is therefore to plead the urgent necessity for efficient splinting by relatively simple apparatus in the earliest possible stage; for effective co-ordination between the surgeon and the dental surgeon; and an emphatic protest against the notion, apparently commonly current, that the treatment of bony injuries to the jaws is a glorified species of orthodontics.

Mr. F. J. BENNETT.

I would like to make one remark in connexion with these interesting cases, that is, that all connected with this war work on jaws should be careful to preserve every tooth which has been taken out, so that in peace times we may be able to examine them carefully. Many members will remember Mr. Tomes's remarkable specimen showing healing of the pulp after severe injury. Amongst the numerous cases of fractured jaws there must be some cases in which fractured teeth have healed to some extent. Therefore, if members will preserve all teeth which are taken out, and either keep them themselves or give them to someone who will make a microscopical examination of them, I feel sure we shall learn something more about the anatomy of the teeth.

Mr. WARWICK JAMES.

In reference to the remarks which have been made upon the contraction of scar tissue, an expedient which I have found valuable may be of interest to some members. This consists of the pressure obtained from a length of rubber tubing introduced into the mouth in such a manner as to bring the pressure upon the contracted tissues. Quite thin tubing can be introduced at first, causing gentle pressure, heavier tubing being introduced later. The pressure is obtained by arranging the tubing in a U form, and may be increased by tying the two free ends together to form an oval. In the case of a man injured by shrapnel, who dribbled very badly on account of the fixation of the angle of his mouth to the mandible, the tubing was introduced into the sulcus between the cheek and the maxilla above, and over the scar between the mandible and the cheek below. The man received immediate benefit from the presence of the tubing, and after a week or so the dribbling was completely controlled. The amount of dribbling in this case was so marked that a notice that was placed in the ward requesting patients not to spit was fixed by one of the other patients, attempting to be humorous, upon the man's back. My chief experience in stretching scar tissue in this manner has been in cases of hare-lip and cleft palate.

Mr. PEYTON BALY.

I have only had one case of the kind under discussion since the War started. That man had a bullet wound on the right side of the mandible, and it came out in the bicuspid region on the left side; it took away part of the cheek, horizontal ramus, and a portion of the lip. He was admitted to hospital with his tongue hanging down his neck. Plastic operations were done and the cheek and lip restored, but I was not called in until everything was healed up. Then I could only get two fingers into his mouth horizontally; I could not get an ordinary impression tray in. He had no teeth in the upper jaw, and a right canine and lateral only in the lower jaw. I fitted bands to these two teeth and a vulcanite base-plate on the right. I made up a bite block with a piece of thick gold wire attached to the base on the right, going round and finishing in a loop on the left. I got my bite with a large block, distending the cheek as much as possible on the left, and made up my case with six front teeth on the front of the upper plate, the

molars and bicuspid on the right inside the palate, the lower bicuspid and molars being placed well outside the ridge, as the mandible was much displaced to the left. I gave him a decent articulation on the right. I made no attempt at that stage to replace the malposition. There was no bone on the right horizontal ramus. On the left I gave him a vulcanite block on the upper plate, and a corresponding large portion of vulcanite on the lower, pushing his cheek out. This was united to the right side by the gold wire which was used in getting the bite. I intended to add vulcanite gradually on to that block, so as to push the jaw over slowly to the right, but within three days he was discharged from hospital, and I have not seen him since. I was not called in until the general surgeon had done his part, and as soon as the apparatus was in the patient was sent down to his dépôt.

Mr. F. J. PEARCE.

I should like to express our sincere thanks to our French confrères. To those of us who visited France they gave a most hearty welcome, and put themselves to considerable inconvenience to show us everything there was to be seen. We cannot be too grateful to them for that courtesy.

With regard to the opening of the discussion by Mr. Lewin Payne, I think he has brought out very admirably the main points which one would wish to hear discussed. His classification, although possibly it may not include all cases, does, at any rate, represent a working basis for recording them.

I think it would be very unfortunate if some of Mr. Colyer's remarks were accepted as representing the views of the average dental surgeon in regard to these cases. One recognizes that Mr. Colyer has done an enormous amount of good work in cases which are extremely difficult, owing to there having been so much delay in treatment. But I think he did not make it sufficiently clear in his remarks that the points which he laid down for treatment were chiefly applicable to delayed cases. For instance, extraction of teeth on either side of the fracture is a point with which I think the majority of us can hardly agree. We agree with him when he says the tooth or teeth in the line of fracture should be removed; that is what those of us with experience of fractures, even before the War, have been in the habit of doing. In delayed cases extraction will make the treatment very much shorter, and there may be good union; but in most of the cases I have seen in which that has

been done there is a good deal of loss of function. Surely our aim is not solely the making of these men fit from the military point of view; we have to think of the man as having undergone serious injury, and to consider, not only his fighting possibilities, but also his future life, which, it seems to me, is a point Mr. Colyer has not sufficiently taken into consideration.

The four points with which Mr. Lewin Payne concluded should, I think, be emphasized—namely, all cases of jaw injury should receive the assistance of a dental surgeon as soon after the injury as possible. This cannot be too much insisted upon, especially at a meeting which includes members of the Surgical Section. The second point is, that the treatment should be begun as soon after the injury as possible. Those who have seen hundreds of these cases under treatment are convinced that it can be commenced much earlier than the average dentist might have considered possible. Mr. Payne's point as to the segregation of these cases into special hospitals, or special departments of hospitals, is one which we, especially as members of the Surgical Section are present, wish to emphasize to the authorities. We have done our best, as dentists, to impress upon the authorities the need of this. It may be thought we are getting these cases in the delayed form because the authorities were not warned, but that is not the case. The heads of both the Navy and Army Departments were warned of the need before any soldiers left this country. We also promised them that we would place the whole of our services at their disposal to ensure an efficient service for this kind of case. But, though we have reiterated the necessity from time to time, very little has been done, and I think the authorities are extremely to blame for these cases reaching us without treatment six or eight months after the injury.

MR. J. G. TURNER.

I have had some little experience of these cases in the present War. The sepsis in some of the cases is remarkable, worse than I have ever seen. I find Dakin's solution extremely useful; and chloramine (now termed "tolamine"), another of Dakin's productions, has been very well spoken of by a man from the Dardanelles for these jaw cases. Whether one should attempt much in the presence of such sepsis is an important point. But Claude Martin and his pupils were never frightened by a large amount of suppuration in dealing with bone cases in the jaws, screwing flanges into bone, and so on. They expected

prolonged suppuration. One wonders whether one dare risk it. A man who has been exhausted by fighting had better not be treated in that way; it seems better to make use of antiseptics to kill off as much as we can; Dakin's hypochlorite solution does not kill the tissues at the same time.

The various classifications of fractures we have agreed to fairly well. A fracture without loss of bone can be efficiently dealt with provided there are a fair number of teeth left.

Pace Mr. Pearce's fears as to public opinion, I think Mr. Colyer's suggestion to treat delayed fractures by extraction on the line of fracture has proved itself. When a fracture occurs behind all the teeth, I think a removable cradle splint should be used.

I agree with other speakers as to the difficulty of using cemented splints in the kind of mouths we see, especially when there is the possibility of having to take off a splint to remove a root.

Where there is but small loss of bone, but including the whole depth of the mandible, I believe, as Mr. Stanley Mummery said, there is a very good possibility of fragments of bone and osteogenetic tissue being left, and that is all that is wanted; bone will be regenerated. If the skiagram shows such fragments, we should fix the two main fragments apart, and so give the scattered fragments a better chance of regenerating the bone. If there are no such fragments, then putting them together and, later, stretching, may lead to an equally good result. It may also be possible to turn down into the gap a piece of bone from the patient's mandible, hingeing it on a vital union of periosteum. There we need, by a cap splint on the teeth, to make the two portions immovable. Where there was large loss of bone, but plenty of soft parts remaining, Claude Martin used large masses of vulcanite, replacing a temporary apparatus of the same kind flanged on to the bone fragments. The masses of vulcanite used for temporary work should be solid; there should be no attempt to drain through the vulcanite, as originally advocated. The temporary apparatus should, if possible, be fixed to the fragments of bone. Where there is large loss of tissue, there is less to be done, and one must look to the surgeon for plastic operations. I think we may be blaming the surgeon too much in calling attention to the contracted mouths which we see later; there may not have been the possibility of doing a decent plastic operation.

Bone-grafting seems to open up a useful field. I should like to speculate on one or two possibilities. The tissue from which bone is renewed is not periosteum, but bone cells under the periosteum and in

the medullary portion. In the lower jaw one can frequently see the medullary portion renewing bone. For instance, the root of a septic tooth, or of an implanted tooth, is absorbed. On taking such a tooth out, one often brings with it a little mass of new bone which has grown into the absorbed hollow of the root. New bone may be deposited on the cortical side of the periosteum to a considerable extent. These cells are delicate. If we are going to make a graft, it seems wrong to pick out the cortical hard bone and dovetail it into other hard bone; there is not the blood supply, nor is the cortex the part which will be penetrated easiest, or which will send out the bone cells to the greatest extent. We should take as much of the medullary portion as possible, and fit it in loosely, for fear that the pressure may kill the cells. So here we need a good intra-buccal splint to allow the implanted bone to lie easily in the cavity. Perhaps the amount of bone should be small, so that the nourishing lymph may have access to it.

The case in which Sir Frederic Eve embedded phosphor bronze wire leads one to hope that it might be a permanence. But I think that is very unlikely, and if it is not to be so regarded, the line of temporary treatment is not to allow contractions to take place into so small a bulk as the phosphor bronze wire indicates. Large masses are what we try to use, so as to keep space for a permanent apparatus. All metals let into bones which are movable are apt subsequently to work loose. It is only where there is no movement that you may expect the bone to overlap the metal, or even, as has happened, grow through the perforations in that metal.

A point I would emphasize refers to cicatrization. Once you have stretched the cicatrix, a return can be prevented by the exertion of a small amount of force, but it must be applied daily, otherwise you will soon learn that the cicatrix is going back. It is not the case that once you have stretched, the matter can safely be left.

MR. LEWIN PAYNE (in reply).

In concluding this debate, I shall only attempt to refer to a few of the points which have been raised.

As Dr. Hayes explained in his opening remarks, no new pathological problems are presented by the cases we have been discussing, but the difficulties to be contended with are those relative to the complexity and variety of the deformities. These problems are essentially the work of the dental surgeon, and, to repeat the statement of

Sir Frederic Eve, the assistance of the surgeon is required only exceptionally, if the treatment by the dental surgeon has been carried out skilfully from the outset of the case.

Mr. Colyer will hardly expect me to agree with all the details of his treatment. I should lay more stress upon the retention of the individual teeth, also upon restoring the teeth to the position of occlusion which is normal to them, and I agree with the contention of Mr. Hern, and of others, that the removal of teeth on either side of each fracture is undesirable in most cases. In regard to these views we must remember, however, that Mr. Colyer has been dealing largely with neglected cases, which should be placed in a different category from those patients who have been recently injured.

Mr. Turner urges that it is desirable to sacrifice teeth for the sake of getting union. I would ask him to make a journey to France, where patients are grouped together in hundreds, and there he will see how much better results can be secured by retaining the teeth and restoring normal occlusion. Moreover, there are cases, as Mr. Norman Bennett has shown, in which it is better to have fibrous union with good occlusion rather than bony union with bad occlusion.

Mr. William Hern's work in connexion with the treatment of fractures of the jaw extends over a long period, and his opinion is of great value to us. I strongly support his opinion as to retaining fragments of living bone. Mr. Hopson's case very well illustrates the serious results of getting rid of loose pieces of bone at a premature stage.

In order to illustrate the importance of early dental treatment, I would like to show pictures of two cases which in many respects are similar in character. They are of the type I have referred to under Group 4—i.e., two or more fractures of the mandible involving loss of substance on both sides. In each instance the bullet entered the mandible on the left side, carrying away two or three teeth with the corresponding bone below them, and found exit on the right side, where smaller destruction occurred.

Here are the models of the first patient (*see fig. 9, p. 74*). He was wounded on May 19, 1915. No attempt had been made to correct the displacements of his mandible until he came to see me more than three months later. During the interval several pieces of bone were removed, and some teeth had been extracted. The fragments were loose, and there was no true occlusion of any of the teeth. It has been possible to restore the position of the remaining teeth; they are now in occlusion, and bony union has occurred in the right second premolar region, but the

gap on the left side, between the right central incisor and the left second molar, remains ununited. This patient, at the end of nine months, has only fibrous union on the left side, whereas I believe he might have had bony union on both sides of the jaw if an interdental splint had been employed at an earlier date.

I want to contrast this case with that of an officer who came to me soon after he was wounded. He was shot by a bullet on June 11, 1915. I saw him thirteen days later. The radiogram (*see* fig. 6, p. 73) shows the condition on his arrival in London. The damage and the position of the teeth were similar to the case I have just referred to. When the patient was lying in bed the anterior fragment fell over towards the back of the mouth. An open cradle splint was ligatured to the maxilla and mandible a week or so after his arrival in London. The maxillary ligatures were removed after two days. Subsequently he was treated with a closed cradle splint, with the teeth in occlusion, and after eight weeks he was able to use a modified form of denture with intermaxillary flanges which retained the fragments in their relative position. With this he could manage to eat soft food, and in October (a month later) he was passed by the Medical Board for light duty. Last month, the jaw having united, he returned to full duty, ready to go abroad again (*see* fig. 7, p. 74).

In the first case the patient, after ten months, is still unfit; in the second he is well and able to return to active service.

It has been shown in this discussion that if the dental part of the treatment is undertaken early enough and in a manner that has been suggested by many speakers, the country will be saved thousands of pounds, the patients will be made useful citizens for the future, and in a very large number of cases they may again become soldiers ready to return to the Front.

Mr. T. S. CARTER (communication by letter).

Wire suturing of the jaws, by at once causing fixation, is quickly followed by osseous union. I am in favour of silver-plated copper wire, No. 19 B.W.G., and a special key for twisting it up, also flexible needles for returning the wire. The drill must not be revolved too rapidly or advanced too quickly, or necrosis may be caused by heating. A small rubber tube to protect the lip is put over the twisted ends of the wire before being turned down, or a thin strip of rubber dam may be wound round them and the two ends tied as a finish.

Odontological Section.

President—Mr. H. BALDWIN.

(May 22, 1916.)

On the Structure and Arrangement of the Enamel Prisms, especially as shown in the Enamel of the Elephant.

By J. HOWARD MUMMERY, D.Sc.Penn., M.R.C.S.

It might, perhaps, be thought that the subject of the ultimate structure of the enamel prisms had been so fully investigated that there was little or nothing more to be learned about it. There are, however, still some undecided points with regard to this subject upon which I hope to throw some further light in the following paper.

In 1905, E. Smreker published a paper [4] in which he asserted that the majority of the prisms in human enamel are not round or polygonal in shape in transverse section, but have an arched form, owing to the prisms being longitudinally grooved and fitting into one another; he also described feather-like processes from the prisms, and a system of connecting processes or bridges between them. These observations were confirmed by Professor von Ebner, who deals with them at some length in a paper published in the same year (1905) [3]. These conclusions have not, however, been generally accepted or confirmed by others, nor have they been referred to in recent text-books, no reference to either of these papers appearing in the new edition of Tomes's "Dental Anatomy."

Professor Walkhoff and others look upon them as due to errors of observation, and consider the arcade form of the prism to be a false appearance. An investigation, however, upon the microscopic structure of the enamel of the elephant which I have recently undertaken has,

I think, thrown fresh light on the subject, and tends to corroborate the description of the ultimate structure of the enamel prism as given by Smreker and von Ebner.

The peculiar structure of the enamel of the elephant reminded me of the illustrations in Professor von Ebner's paper, which I had always found difficult to understand, never having then seen appearances in human enamel such as he has described. A careful study of this paper makes it evident that we have in the enamel of the elephant a structure similar to that described by Smreker as seen in human enamel. In the elephant it is seen on a larger scale, and is more generally shown throughout. This form of modification of the prism was first described by E. Smreker in 1905, and his work is quoted by von Ebner in his paper on enamel, where he expresses surprise that Smreker's observations have not been more generally recognized, the reason for this probably being the great difficulties of observation in human enamel

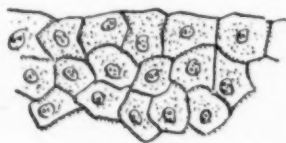


FIG. 1.

Prickle-cell layer of pavement epithelium.

and the necessity of procuring very thin ground sections. Smreker stated that in thoroughly polished sections, either impregnated with silver nitrate or unstained, the cross-section of the prism was not always rounded or hexagonal, but in many places showed a predominant form which reminds one of the arrangement of the cells in a pavement epithelium. He says: "As in the wing or prickle-cell layer of a pavement epithelium, the cells show an arched surface and an under surface furnished with facets, which are conditioned by the arched surface of the underlying cells, so do the enamel prisms also abundantly show an arched surface on one side which is arranged over a more or less concave surface, which is dependent upon the arched surfaces of neighbouring prisms" (fig. 1 in text). He describes the convex surface as being directed almost without exception towards the dentine. If the prisms lie in regular rows behind one another they show single grooves on the posterior surfaces; if in alternating or irregular rows they show

two or more grooves at the back. He confirmed these observations with teased preparations, and von Ebner also investigated the enamel of the wild boar and of the calf, which gave indications of a similar structure. von Ebner, when he first read Smreker's paper, thought as I did, that this appearance was due to the sections being viewed obliquely, but teased preparations showed him that it was an actual structural condition. As I shall presently describe, I have found in successful teased preparations of elephant enamel both the single and the double grooves as well as an arched shape of the broken-up prisms in transverse section.

The authors referred to describe wing processes of the prisms (the "Flügelfortsätze" of von Ebner), connecting processes between the prisms, which are described as intercolumnar bridges, and a needle-like splitting of the prisms.

Wing Processes of the Prisms.—In teased preparations of most mammalian enamels one finds here and there the so-called wing processes (Plate, figs. 12, 13 and 14). These are lateral projections from the prisms, appearing sometimes as projecting, obliquely directed fibres or processes, sometimes showing a zigzag splitting like an edge of fractured glass. They can be seen extending all across the prisms as well as projecting from its edges, and von Ebner has figured them in human enamel as enveloping the concavo-convex prisms like a sheath, and projecting on either side. These wing processes are seen not only in forming enamel but also in the completed tissue. Here and there in teased preparations membranous expansions are seen attached to the prisms, and, although von Ebner does not think these are identical with the wing processes, an examination of teased preparations of marsupial enamel has furnished me with considerable evidence that they are identical, as I hope to show presently.

Intercolumnar Bridges (Plate, fig. 9).—Connecting processes between neighbouring prisms are seen in human enamel, and have been described by Leon Williams. The term "intercolumnar bridges" is suggested by von Ebner for these connecting processes, and I have adopted it in the present paper.

Needle-like Splitting of the Enamel Prisms (Plate, fig. 12).—In all teased preparations of mammalian enamel that I have examined the prisms are seen to terminate in oblique, needle-like points at one end, and sometimes at both ends. This splitting is evidently, in most cases, not at the same angle as the projections of the wing processes, and is apparently due to the longitudinal fibrillation of the prisms so evident in the forming enamel of marsupials, and described by Andrews and

Leon Williams in human enamel. The direction of these needle-like projections would appear to suggest that they are due to calcification in the direct prolongations of the Tomes's processes of the ameloblasts, which are produced into the longitudinal system of the organic fibres forming the foundation of enamel; the transverse membranes above described—and, as I consider, the wing processes also—forming the fibrillar material which represents the other portion of the organic matrix, both being eventually almost lost to view in the dense calcification of the finished tissue.

I have dwelt upon this investigation of Smreker and von Ebner in order to make clear the explanation of the structure of the enamel of the elephant, and to show that, on a larger scale, the appearances above described (owing to the larger size of the prisms and the more general existence of this mode of arrangement) are more easily demonstrated in the elephant than in human enamel. This study helps to explain more effectually some appearances and, I think, to suggest a different explanation of certain disputed points.

In studying the wing processes I have made use of teased preparations of marsupial enamel, and have found in the kangaroo (*Macropus rufus*) appearances which, I think, throw considerable light on the nature of these processes. In these animals the processes are much more apparent than in any other enamel I have examined, and have a more feathery appearance—they often pass right across groups of prisms and appear to show a transition into membrane-like expansions. Marsupial enamel appears to give us the key to the formation of other mammalian enamels; it is less perfectly calcified, at all events, in early stages, and the structure is more easily made out than in a tissue which is very rapidly calcified, and in which the early stages of development are soon obscured by the densely deposited lime salts. I have not been able, however, to detect in marsupials the curious interlocking of the prisms seen in elephant and other mammalian enamels; the prisms appear to have the usually described polygonal outlines, and I could detect no longitudinal grooving. It may be that the interlocked prisms indicate a more highly developed and specialized structure of the enamel.

While many investigations of the anatomy and macroscopical structure of the molars of the elephant have from time to time appeared, I have not met with any giving details of the microscopic structure of the enamel. Professor Owen, in his "Odontography," refers to it very briefly. He says: "The fibres of the enamel present a minutely jagged or crenate outline, but are not crossed by transverse

lines. Their diameter is $\frac{1}{3000}$ in. They are much less wavy than in the human teeth." As he speaks of them as not being crossed by transverse lines he is evidently speaking of the longitudinal view of the prisms, which have a distinctly crenated outline. Cross-striation of the prisms is distinctly to be seen, however, in teased preparations of elephant enamel as shown in several of my preparations.

In the adult animal the enamel is confined to the molar teeth, but the permanent tusks when erupted have a thin coating of enamel, which is at first entirely covered with cementum even at the tip, as shown in the first instance by Professor W. D. Miller, who made a prolonged and careful study of the anatomy and pathology of the teeth of the elephant. A section made at right angles to the vertical surface [2] of the lamellæ of the molar shows alternate bands of cementum, enamel, and dentine, and as described in a paper by Professor Miller and Dr. Dieck, the

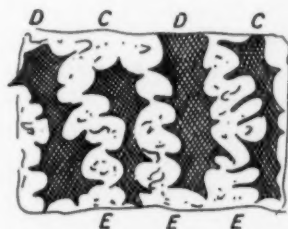


FIG. 2.

Transverse section of molar of elephant, showing the thorn-like processes of dentine and cementum. E, enamel; D, dentine; C, cementum. ($\times 2$)

interlocking of the tissues is very complete owing to a peculiar arrangement which they were the first to describe. Thorn-like processes or projections of the cementum extend into the enamel on one side, and similar processes extend into it from the dentine on the other side (fig. 2 in text). As Miller points out, a similar structure is found in the compound molar of the wart-hog (*Phacochoerus*) and also in the teeth of the hippopotamus, but in these animals it is only the cementum and not the dentine which has these prolongations into the enamel. This interlocking of the tissues would probably be of assistance in enabling the molar of the elephant to stand the enormous strain to which it is subjected. Such an arrangement is fairly common in herbivora.

On examining under the microscope a transverse section of the enamel of the Indian elephant, which also included the neighbouring

areas of dentine and cementum, I was struck with the great size of the enamel prisms. A measurement showed an average diameter in transverse section of $10\ \mu$, while the prisms in human enamel have a diameter of from $4\ \mu$ to $5\ \mu$. Such a section shows prisms both in their transverse and longitudinal aspect, the former predominating, and the transversely cut prisms appear to overlap one another like tiles on a roof or the scales of a fish, but no prisms having a rounded or polygonal form can be seen over the whole surface of a large section. The most obvious explanation of this appearance would be that they are rounded or polygonal prisms which are viewed obliquely, appearing to be over one another, either one or two overlapping the one in front. In this case, however, the most obvious interpretation is not the true one, except to a very limited degree, as I shall hope to show presently, for the great majority of the prisms have a concavo-convex figure in cross-section, and in longitudinal section show a convex anterior aspect, directed towards the dentine, and a single or double groove traversing the length of the prism on the posterior aspect (Plate, figs. 1, 2 and 3). This is clearly seen in fragments of enamel teased in glycerine. When von Ebner first read Smreker's paper, he thought, as I did, that this appearance was caused by the sections being viewed obliquely, but the teased preparations show that it is a real structural condition.

In successfully teased preparations of elephant enamel I have found both the single and the double grooves very evident, as well as an arched shape of broken-up prisms in transverse section. In human enamel I have frequently found the wing-like lateral expansions in teased preparations, and have also in many cases seen (in sections) the arcade form of the prisms seen in the elephant, but I have not been successful in finding grooved longitudinal prisms which I could clearly describe as such. I have not, however, made a very extended examination of human enamel for this purpose. I think it is certain that this form of the prism is not so general in human as it is in elephant enamel, since an examination of many sections of human enamel shows the cross-striation, which is more marked than in the elephant, to be continuous across neighbouring prisms not showing any curvature, and we see many cross-sections of human enamel which show the usually described polygonal outline.

In fig. 3 in text I have shown a transverse section of elephant's enamel, and it is plainly seen that the prisms do not show the usually described form but have a rounded anterior and a concave posterior extremity, giving a festooned appearance to the surface, as if made up

of overlapping tiles. These sections when placed immediately behind one another in a single row have a single concavity at the back; the more usual arrangement, however, is alternate, and the prisms have a double concave posterior margin. It is difficult to convince oneself at first that the whole appearance is not due to an oblique view of regular polygons, but a search over a large area shows similar forms everywhere. In many parts of the sections, not only have the prisms this tile-like appearance, but they are seen to have a serrated margin, and they also appear as if grooved or ridged on the surface of the section, many prisms having a singular resemblance to the frond of a maidenhair



FIG. 3.

Transverse section of the enamel of the Indian elephant. ($\times 800$.)

fern (fig. 4 in text). The convexity of the prism in the elephant seems always to be directed towards the dentine and not towards the cementum on the opposite side of the section. In longitudinal sections the prisms appear wavy, and in many places show grooves and overlapping feathered margins (fig. 5 in text).

By means of teased preparations of elephant enamel I endeavoured to ascertain the actual meaning of these appearances in sections. The exposed surface of the enamel was scraped with a sharp knife and the little detached fragments broken up with needles in glycerine, water, or

Farrant's solution on a microscope slide. Fragments of enamel showing rows of prisms in a longitudinal aspect demonstrated that the columns were united by processes which are arranged in rows between them, giving them an appearance as if sewn together with rather thick and irregular stitches—these, the intercolumnar bridges of von Ebner and the processes between the rods of Leon Williams, are very distinct, and, as noticed above, are not quite regular in arrangement (Plate, fig. 9, and fig. 7 in text). Separate groups of enamel prisms also showed very distinctly the single or double groove at the back, and I was fortunate enough in a few instances to see a prism broken in two at the summit of the groove, showing how the typical appearance in transverse section was produced (Plate, fig. 3). In the double-grooved prisms there is a distinct ridge between the two grooves, which appears to be continued into the ridges seen traversing the transverse section in many places. The single-grooved prisms have a very distinct ridge on either side which is of an appreciable breadth, and little knobs or projections can be seen along the ridge, apparently caused by the broken bridges connecting the ridge with the sides and convexities of the neighbouring prisms. A few fragments of prisms broken up transversely show various shapes, from a single concave to a double concave posterior margin and with varying depths of the concavities, so that in some of them the prisms have become flattened out and only show very narrow grooves. Some fragments of enamel are also seen, which give very definite information as to structure. These pieces show groups of prisms in transverse or oblique section, and at one margin the grooved edges project from the surface and at (b) in Plate, fig. 10, the whole of one of these arched prisms is seen *in situ* with its concavity projecting freely from the surrounding prisms.

The grooving of the prisms is seen in sections as well as in teased preparations (Plate, fig. 8, and fig. 5 in text), and in many parts the longitudinal sections show the overlapping wing or feather processes (Plate, fig. 7).

The photograph (fig. 6 in text) shows, I think, very beautifully the actual arrangement of the double concave prisms. It was photographed at a crack in the enamel, and the prisms are seen both in longitudinal and transverse section, and the little shining points, apparently due to the broken connecting bridges, are also visible. A comparison of this figure with fig. 3, Plate, from the teased preparation will, I think, fully confirm the view of the structure of the prisms detailed above. From this photograph and from many preparations it would appear



FIG. 4.

Elephant enamel (transverse section). ($\times 800$.)



FIG. 5.

Grooves and wing processes in the enamel of the elephant. Ground longitudinal section. ($\times 500$.)

that the prisms of the enamel are arranged, not in direct lines from the dentine, but that, independently of all the curves and intercrossing, they maintain a slope towards the dentine which it is very difficult to follow out or define, and may possibly be connected with the uniform curve of the enamel in all directions around the dentine.

Connecting processes between the neighbouring columns of prisms are seen in human enamel, and have been described by Leon Williams; they are very evident in the elephant, and are particularly well shown in the photograph (fig. 7 in the text). These processes are seen to pass across the interprismatic substance and form connexions with the neighbouring columns. von Ebner says that they are not seen in transverse sections of enamel, but in many places in the elephant they are very distinct (Plate, fig. 6), and in one of my sections of human enamel bridges are also quite clearly seen in transverse section. In fig. 7 (in the text) the bridges are seen as very clearly defined connecting processes, and it will be noticed that they are somewhat irregular in form, as are the connecting or wing processes of the prisms (Plate, figs. 13 and 14). von Ebner, however, in describing human enamel, says that these bridges never show uneven connexions between neighbouring prisms, but banded obliquely directed plates, and therefore they do not conform to the wing processes, but they certainly appear in the elephant to form uneven connexions.

The membrane-like expansions sometimes seen in teased specimens of human enamel von Ebner does not consider to be identical with the wing processes, but in teased preparations of marsupial enamel these membrane-like expansions are much larger than in other mammalian enamels; they seem to take the place of the wing processes in many places, and show a very marked fibrillation (Plate, figs. 12 and 13). That these membranous expansions and wing processes in man and the elephant are calcified is shown by polarized light, and the very irregular zigzag splitting of the wing processes is, I think, probably due to the fracture of these membranous layers in teased preparations. From this comparison with marsupial enamel I am inclined to think that wing processes and membranous layers are one and the same thing, but in teased preparations of the enamel of higher mammalia the expansions are more completely calcified than in marsupials, and their remains are only seen as the wing processes of the prisms.

While, then, the needle-like splitting of the enamel in teased preparations is, I think, due to the longitudinal fibrillation of enamel, as I suggested above, the membrane-like fibrillar expansions, the wing

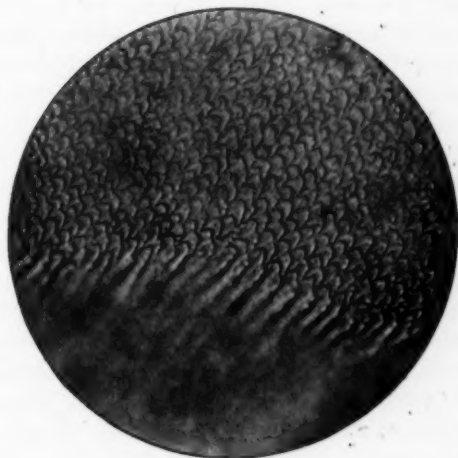


FIG. 6.

Enamel of elephant photographed at a crack, showing prisms in both longitudinal and transverse sections. ($\times 400$.)

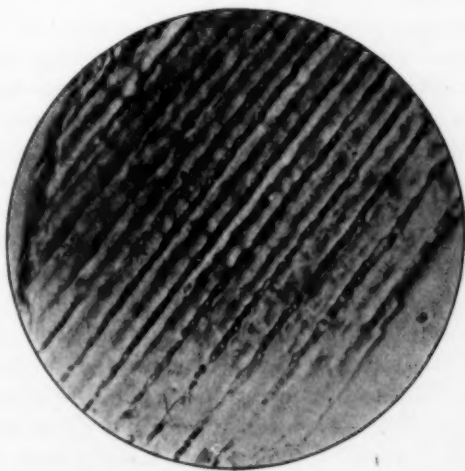


FIG. 7.

The intercolumnar bridges (from a teased preparation of elephant enamel). ($\times 600$.)

processes and the bridges, I am inclined to consider as all due to the transverse fibres of the enamel matrix, which are very apparent in the forming enamel of marsupials [5], where the teased preparations break up into laminae. These transverse fibrillar layers must become incorporated into the substance of the calcified prism, or it would scarcely be possible to account for the appearance of intercolumnar bridges in transverse section; they evidently form a portion of the solid prism (Plate, fig. 6). These grooved prisms are not in direct contact with one another, being separated by the interprismatic substance, which has flowed around them as it were, cementing together and compacting the whole tissue. The interprismatic substance is, however, crossed by the bridges which effect a direct communication between the prisms. Walkhoff holds that there is no interprismatic substance, that this cement substance is simply the cortical or external layer of the prisms, and that calcification in the enamel is a centrifugal process leading to a complete fusion of its whole substance. Mr. Charles Tomes considers that calcification of the enamel takes place centripetally, calcification proceeding from the periphery to the centre of the prism, and he would probably also consider that there is no true interprismatic substance. Leon Williams considers the interprismatic substance to be a separate material formed independently of the prisms, and my own investigations on the enamel of marsupials, I think, tend to confirm this view.

A further confirmation of the separate formation and distinct nature of the interprismatic substance is given in a recently published paper by the late Dr. Black [6] in which he describes an endemic pathological condition of the teeth occurring in certain districts of the Rocky Mountains. This condition he calls "mottled teeth," from the alternate pigmentation and bleaching of the teeth seen in this affection. This pathological condition affects only the interprismatic substance and not the enamel prisms; the work of calcification appears to be reversed and the last-formed part of the enamel, the interprismatic substance, is removed by the action of some as yet unknown agent which has no effect upon the prisms. This remarkable complaint, investigated on the spot by Dr. Black and by means of a very carefully prepared series of microscopical sections, lends the greatest confirmation to the view that the prisms and the interprismatic substance are separate structures. The question then arises, How are they distinct from one another—is the cement substance only a persistently less completely calcified element of the enamel, or is it in some way of different composition?

Leon Williams stated that this substance is formed independently

of the prisms, and in my own investigations on calcification I have shown that large calcospherites in all conditions of coalescence are seen surrounding and enveloping the forming enamel prisms which are seen, as Leon Williams describes, to be built up of little blocks of uniform size. The mode of formation is thus distinct in the two calcified elements of the formed enamel, the prismatic calcification being deposited in regular, uniform bodies due to the coalescence of granules, the interprismatic substance by the fusion and disintegration of large calcospherites.

If we accept these observations of Smreker and von Ebner on human enamel as correct—and they appear to be confirmed by my own on the enamel of the elephant—the structure and arrangement of the prisms are rather more complicated than we have usually considered them. We have first the laying down of the calcifying material in granules within the fibres of the Tomes's processes of the ameloblasts described by Andrews, Tomes, and Leon Williams, which is so evident in marsupials. These granules become consolidated into blocks of the enamel substance arranged in regular order in a longitudinal series and giving rise to the appearance of cross-striation. We have also a transverse striation between and across the prisms due to a fibrillar organic structure, also derived from the epithelial enamel organ, but not seen in fully calcified enamel. These are seen in the closely applied laminae observed in the forming enamel of marsupials; these laminae I am disposed to consider form the wing processes which are seen in both calcifying and completed enamel. The laminae become incorporated in the calcified substance of both prisms and cement substance as calcification advances, and their incorporated fibres form the connecting bridges seen in both longitudinal and transverse sections.

While in human enamel many prisms show the usually described polygonal or hexagonal forms, the majority, if not all, in the elephant possess a more or less concavo-convex figure, being grooved longitudinally, the contiguous prisms fitting into the grooves and forming a closely compacted tissue; the inter-prismatic substance, small in quantity, in fully calcified enamel, being crossed by the intercolumnar bridges. Grooves and feather or wing processes are visible in fully formed mammalian enamel. This interlocking of the prisms would appear to be an effectual arrangement to resist the effects of strain, and the immense pressure brought to bear on the molars of the elephant may be a reason why this arrangement is so general in the enamel of this animal.

The points in this investigation in which I find myself not able quite to agree with Professor von Ebner are the following:—

(1) I find inter-columnar bridges distinctly visible in transverse sections.

(2) I am inclined to consider the wing processes and the membrane-like fibrillar expansions as identical, the latter being very evident in marsupial enamel, where the transition from these membranes to wing processes appears to be distinctly shown.

(3) The vertical needle-like splitting does not appear to be due to the wing processes but to the longitudinal fibrillation of the enamel

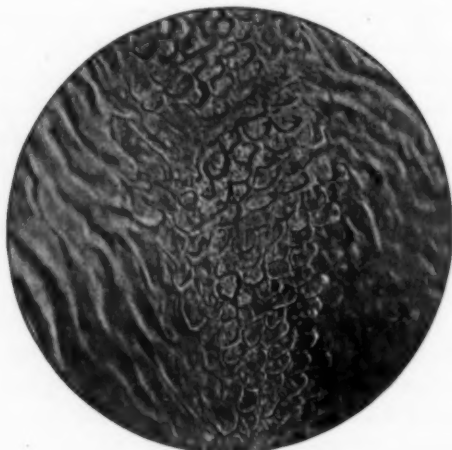


FIG. 8.

Transverse section of human enamel, showing arcade forms. ($\times 750$.)

prisms, due to the prolongations of the Tomes's processes of the ameloblasts, as in marsupials the fibrillar projections from the prisms are often at right angles to the direction of the needle-like splitting.

(4) The inter-columnar bridges appear to be due to the transverse membranous expansions and the wing-like projections.

I do not feel justified, however, in affirming these points as to the enamels examined by Professor von Ebner, but only with regard to those mentioned in this paper, and hope by fresh preparations of human enamel further to confirm his main contentions. I would refer those who are interested in this subject to the paper by Professor von Ebner mentioned above, which gives a very full consideration not only of the form and arrangement of the prisms in man but also of the

action of polarized light on the enamel prisms, the permeation of the enamel by stains, and other points connected with its structure and development.

Another noticeable fact in the enamel of the elephant is that there is a fairly regular passage of the dentinal tubes into the enamel at short intervals round the margins of the two tissues; these tubes do not, as a rule, penetrate very far, but I have traced several of them (in a section in which the dentine was stained with eosin) to the width of a dozen of the transverse sections of the prisms. Many of the so-called "enamel spindles" are also seen, but they appear to be clearer and have not the coarsely granular appearance usually shown by those in human enamel.

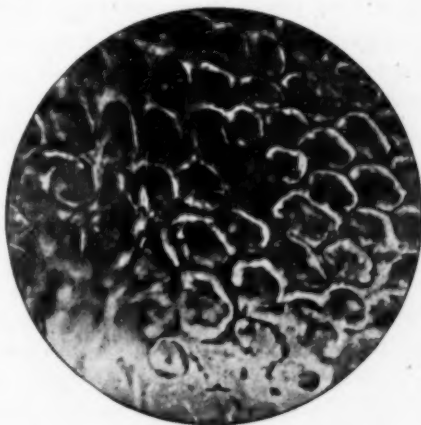


FIG. 9.

As fig. 8, but more highly magnified. ($\times 1,500$.)

I have made some sections of the enamel of the African elephant which show an arrangement of the prisms similar to that seen in the Indian elephant.

Fig. 8 shows the arcade form of the prisms in a transverse section of human enamel, and fig. 9 a similar preparation magnified 1,500 diameters.

I have prepared a set of models in wax and in wood of this peculiar arrangement of the enamel prisms in the elephant, and these demonstrate the special points in this structure probably better than any detailed description. The scale of magnification adopted in making the models is approximately 800 diameters.¹

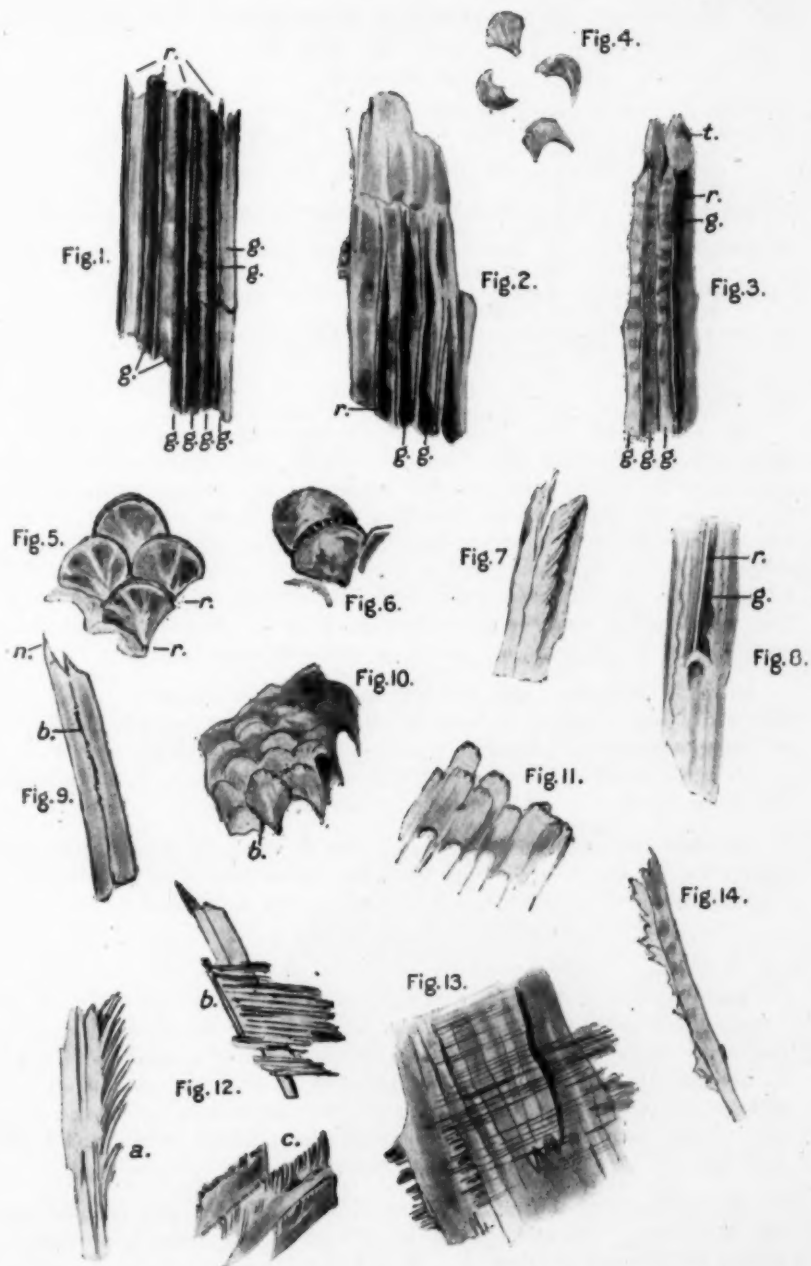
¹ These models were shown at the meeting.

DESCRIPTION OF PLATE.

Drawings from teased preparations of enamel, except figs. 5, 6, 7 and 8, which are from sections.

- FIG. 1.—Double-grooved prisms (elephant). *r*, ridges; *g*, grooves. The ridges are often seen projecting beyond the extremities of fragments.
- FIG. 2.—Single-grooved prisms (elephant). *r*, ridge; *g*, grooves.
- FIG. 3.—Two double-grooved prisms, transverse above (elephant).
- FIG. 4.—Fragments of prisms in transverse fracture (elephant).
- FIG. 5.—Four prisms from a section (elephant) showing surface marking and prominence of the ridge at *r*. Compare with photograph, fig. 4.
- FIG. 6.—From elephant: bridges in transverse section. The interprismatic substance appeared dark and the bridges are very conspicuous as white lines.
- FIG. 7.—Elephant. From a section showing a wing process in the enamel. Compare with photograph, fig. 5.
- FIG. 8.—Elephant. From a section showing ridges and grooves. *r*, ridges; *g*, grooves. Compare with photograph, fig. 5.
- FIG. 9.—Two prisms from elephant, showing needle-splitting and intercolumnar bridges. Compare with photograph, fig. 7.
- FIG. 10.—Fragment of elephant enamel in transverse section. Two entire double concave prisms are seen projecting, with feather edges and intercolumnar bridges.
- FIG. 11.—Fragments of prisms seen obliquely (elephant). Compare with photograph, fig. 6.
- FIG. 12.—Wing processes and membranous layers in *Macropus*, showing needle-splitting, wings and bridging.
- FIG. 13.—Fragment of *Macropus* enamel. Membranous laminae at right angles to direction of prisms.
- FIG. 14.—Wing process, human enamel.
-

The drawings of the teased preparations were made by direct observation under the microscope, and are magnified about 400 diameters; figs. 6 and 7, from sections, are $\times 800$.



MUMMERY: *On the Structure and Arrangement of the Enamel Prisms, especially as shown in the Enamel of the Elephant.*

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DISCUSSION.

The PRESIDENT: The arrangement of the enamel prisms which Mr. Mummery has described seems an ideal one for preventing fracture taking place along the lines of interprismatic substance. It would be impossible for fracture to take place without traversing portions of the prisms themselves, and this is a better arrangement than the hexagonal prisms on the "basaltic column" formation, which used to be imagined to exist in the enamel. Obviously the appearances cannot be due to obliquity of the section, since that would merely result in a lengthening out of the figure representing the cross section of the prism and not in a total alteration.

Mr. A. H. PARROTT: Can Mr. Mummery tell us whether his researches will enable him to give any idea of the microscopical appearances of the enamel prisms on the external surface as distinct from the appearances shown at the amelo-dentinal junction, and whether variations in such appearances will enlighten us as to the incidence of dental caries?

Mr. MUMMERY (in reply): I am not able at the moment to furnish any information on the point, as my recent researches have been made on the enamel prisms of the elephant, and are thus only comparative.

The PRESIDENT: In view of a criticism of Mr. Mummery's researches on "Innervation of Dentine" which has recently appeared in the *Dental Cosmos*, and owing to the fact that some time must elapse before a reply can be made to the criticism, I have asked Mr. Mummery to bring to the meeting the slides of his photomicrographs which best illustrate his conclusions and show them to the members. Mr. Mummery has kindly agreed to this, and I will now ask him to project them on the screen.

The PRESIDENT (after the slides had been shown and described by Mr. Mummery): The slides speak for themselves, and there is no need to defend Mr. Mummery's position. It is unassailable.

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SECTION OF OPHTHALMOLOGY.

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Section of Ophthalmology.

President—Mr. PRIESTLEY SMITH, F.R.C.S.

(November 3, 1915.)

The Death of Mr. George Coats.

THE PRESIDENT: Within the last few minutes I have been told of an event which members of the Section will hear with the greatest and most sincere regret. Mr. George Coats, who, I believe, has been out of health for some time, underwent an operation a few days ago, and he died to-day. I cannot imagine a greater loss to the Society, and to this Section in particular. We shall miss our colleague both as a friend and as a highly valued scientist.

(November 3, 1915.)

Case of Associated Jaw and Lid Movement.

By Captain A. W. ORMOND, F.R.C.S.

CASES of the kind have been shown before, but I consider them rare; this is the first case of the kind which I have had in my own practice. The subject was very ably discussed by Mr. Bishop Harman some time ago, and his paper appeared in vol. xxx of the Ophthalmological Society's *Transactions*. When this boy moves his jaw laterally, the lids are seen to move at the same time. On moving the jaw to the left, the right upper lid is raised, and vice versa. This associated movement of lid and jaw is supposed by Harman to be an atavistic phenomenon, the result of an association such as is found in the lower animals between the muscles which act on the spiracle and those which act on the jaw. The muscles are developed from two layers of muscular tissue—the deep ones for the jaw and the superficial ones for the spiracle—these being associated in the human being with fibres which form the muscles of the face, especially the orbicularis palpebrarum and those which develop into the pterygoid.

(November 3, 1915.)

**Eye Lesions as a Point of Importance in directing Suspicion
to Possible Trypanosome Infection.**

By C. W. DANIELS, M.B., F.R.C.P.

(Communicated and read by LESLIE PATON, F.R.C.S.)

[Mr. PATON: Some little time ago I showed, for Mr. Inman, a case of irido-cyclitis due to infection by trypanosomes. That case brought home to me, and perhaps to many others, the fact that this is a possible cause for irido-cyclitis which has not been sufficiently recognized. I asked Dr. Daniels at that time if he would look into cases of sleeping sickness and ascertain whether it was a common occurrence to find this association. Dr. Daniels has found it to be much commoner than he had supposed, and there have also been many cases in the earlier records in which eye symptoms have been noticed without special attention being called to them. The paper now sent is in response to the request I made to Dr. Daniels.]

THE importance of eye lesions as one of the diagnostic signs of infection with trypanosomes is great, as, unless suspicion be aroused, the nature of the disease is likely to be overlooked, and unless treated energetically upon specific lines, the infection terminates in the fatal stage of sleeping sickness. The earlier the disease is recognized, the greater the probability of recovery. The eye lesions vary and are not alone diagnostic, but the infections with trypanosomes are usually small, and the prolonged and repeated examinations necessary are not likely to be made unless there is ground for suspicion.

The grounds for suspicion are:—

- (1) Continued pyrexia, which does not yield to quinine.
- (2) Enlarged glands, usually cervical.
- (3) An erythematous circinate rash.

Of these, the last is of most value for diagnostic purposes, and is usually, but not always, present in the early stages.

- (4) Ocular symptoms (often absent throughout).
- (5) Orchitis (often absent throughout).
- (6) Delayed pain after deep muscular bruising (very variable).
- (7) Cardiac irritability and weakness.

These signs may not all be present together, and in any case are not to be relied on for diagnosis; but one or more of them, especially (2)

and (3), and to some extent (4), are often enough to arouse suspicion, which must, however, be verified by blood examination and the finding of trypanosomes. In many points there is a similarity to syphilitic infection, and as a positive Wassermann reaction may be obtained in some cases of trypanosome infection where the question of syphilis can be excluded, the correct diagnosis is of great importance and difficulty.

A special reason for viewing eye lesions with suspicion is that they are the first, in some cases, to induce the patient to take a medical opinion; the other signs may not attract attention, even if they are present.

To patients coming from tropical Africa, as all these do, "fever" means malaria, and about this they think they know all that is to be known.

The enlarged glands are neither tender nor painful, and their condition is not usually marked enough to attract attention.

The rash causes no irritation, is usually on a covered part of the body, and is taken for a bruise, which it sometimes resembles. The eye lesions are often painful and always noticeable.

In the subjoined list of cases of Europeans seen in England with this disease, I am desirous for the above reasons of urging the importance of excluding trypanosomiasis as the cause of some of the eye lesions in persons from tropical Africa. I do not mean it to be inferred that this disease is the only, or even the most important, cause of such eye lesions. On the contrary, in several persons from West Africa, with some form of iritis, keratitis, &c., I have not only been able to exclude trypanosomiasis, but have been able to determine the actual causation and verify this exclusion by the effect of treatment and the subsequent course. In my list I include one case of iritis that I consider to be probably due to concomitant syphilitic infection, and I have little doubt that some of the others may be due to that or to other diseases. I give the list as it is, and my readers will all be able to make such deductions as they may think advisable.

I have grouped the cases according to the part of Africa in which the disease was acquired. In the Rhodesian cases, the parasite trypanosome is specially virulent, and is probably a different species. Some consider that the parasites in some districts of Nigeria form a special species. This so far is not proved, but I give the group from all Nigeria separately. The eye lesions are essentially a toxic irido-cyclitis; with this there is a varying amount of keratitis, congestion of the periciliary ring of the sclerotic, conjunctivitis and photophobia, which may be severe or trivial.

Daniels: *Eye Lesions in Trypanosome Infection*

In some cases of infection with trypanosoma of the lower animals, similar eye lesions occur and recur according to treatment.¹

	Cases	Deaths	Eye lesions	Percentage with eye lesions
Rhodesian cases	6	6	5	83.3
Nigerian cases	10	3	4	40.0
Uganda, Congo, and other parts of tropical Africa	16	9	3	18.7
Total ...	32	12	12	37.5

In one of the Rhodesian cases and in three of the Nigerian cases it was the eye lesions that led the patients to seek medical advice, although other symptoms were present but not complained of. As one of these was in South Africa, another in West Africa, one in England and one in the United States of America, and in none was attention directed to the possibility of the toxic agent being trypanosomiasis, I do not consider that I am going too far in stating that the existence of this dangerous source of toxin is not yet sufficiently appreciated.

One of the Uganda cases showed a peculiar form of choroiditis discovered by Mr. Treacher Collins in 1908.

Another sign, not uncommon, in all forms of trypanosomiasis is marked œdema of one or other or both lower eyelids and beneath them. This is not associated with any lesions of the eye, nor with albuminuria, nor with cardiac disease. It may be taken for a similar œdema that is of not uncommon occurrence in another West African infection, namely, that due to *Filaria loa*.

I doubt if the list includes all the eye lesions in our cases, as for many years these were considered to be the result of the large doses of arsenic (as atoxyl) advocated by Koch, which in many cases led to optic atrophy and other ocular lesions. In the lower animals, and also in man, with moderate doses of this drug rapid improvement and disappearance of eye lesions result, although in lower animals (loc. cit.) the lesions recur if the treatment be discontinued.

Mr. Leslie Paton, who has kindly consented to take charge of these notes, has seen with me, and I understand has shown, at the Section of Ophthalmology of the Royal Society of Medicine, one of the cases here tabulated. It was an ordinary case, with very slight photophobia and irido-cyclitis, and nothing specially diagnostic so far as the eye was concerned, but the patient had the rash—"bruises," he called them—enlarged glands and slight pyrexia. In his case, as in others, the eye

¹ *Journ. Trop. Med. and Hyg.*, 1911, xiv, p. 161.

trouble led him to take medical advice; suspicion was aroused, trypanosomes were found in the blood, and he has been under treatment for over fourteen months. He is himself continuing the treatment in the trenches, although there has been no recurrence of the symptoms.

DISCUSSION.

Mr. TREACHER COLLINS: I have been surprised to hear what a large proportion of cases with eye lesions Dr. Daniels has found in trypanosomiasis. I have seen about eight cases of the disease, and in only one of them has there been any eye affection, and that was the instance alluded to in the paper—the patient who had a peculiar form of choroiditis. It is, however, very common to find eye lesions in animals which have trypanosomiasis from inoculation. I have examined pathologically a dog's eye which acquired keratitis after inoculation with trypanosomes, and I hoped to find trypanosomes in that eye. I made smear preparations of the aqueous humour, but could not find any trypanosomes in it, or in microscopical sections of the eye. That experience is in agreement with the statement in the paper just read, that it is probably a toxæmic affection of the eye. The inflammation in the dog's eye was confined to the anterior parts—the iris, ciliary body and cornea. It was especially intense about the root of the iris, and it seemed to spread into the cornea from the periphery. A striking feature about the sections was the presence of a large amount of fibrinous coagula in the anterior chamber, with red blood corpuscles and large and small mononuclear leucocytes entangled in it. The iris also showed diffuse inflammation, but not the posterior part of the uveal tract.

Mr. LESLIE PATON (in reply): There is one point of great interest arising from the paper. Here is a protozoal infection causing irido-cyclitis. It is known that the spirochæte of syphilis is a protozoon. It also causes irido-cyclitis. The work which has been done at Moorfields by Mr. Browning shows that the blood changes in sympathetic ophthalmia are similar in nature to those found in many protozoal infections, such as malaria. This paper suggests a profitable line of research in that direction. May I remind the Section of the extremely interesting paper read at the last meeting of the Section by Mr. Fisher, on the eye lesions in pregnancy, where the probability of the toxins originating from the syncytium was indicated, and the analogy of these cells to protozoa was mentioned? Briefly, the toxins produced by animal parasites seem to have the power of producing this type of eye lesion. I mention the matter because I believe there has recently been a good deal of work done in connexion with the possibility of many cases of pyorrhœa being due to amœbic infection. An amœba has been discovered in many cases of pyorrhœa, and perhaps some cases of irido-cyclitis occurring in pyorrhœa may also prove to be amœbic. The amœbæ are not easy to demonstrate, except on the warm stage.

(November 3, 1915.)

A Fallacy in the Diagnosis of Glioma Retinæ.

By SYDNEY STEPHENSON, C.M.

FROM a clinical standpoint, the expression "pseudo-glioma" possesses a certain convenience, but pathologically, as we all know, it is about as loose and inexact as any term can well be. The conditions that are most commonly mistaken for glioma include metastatic irido-cyclitis and extension retinitis. Gross tubercle of the choroid, too, as pointed out by the late W. A. Brailey,¹ may also lead to confusion, although such cases are admittedly very rare. Lastly, certain congenital anomalies, to which special attention has been directed by E. Treacher Collins,² may be difficult or impossible to diagnose from glioma.

To the foregoing conditions I now add another possible source of confusion, namely, "retinitis exudativa," or "retinitis hæmorrhagica externa," as differentiated by George Coats several years ago in a well-known communication published in the *Royal London Ophthalmic Hospital Reports* of November, 1908.³

I do not for a moment flatter myself that the connexion in question has escaped the attention of some other observers, although, so far as I am aware, nobody has brought it prominently forward as I venture to do on this occasion. For that matter, in a widely quoted communication on "Pseudo-glioma," written by E. Treacher Collins twenty-three years ago,⁴ a series of eleven cases was described, where the appearances that clinically simulated glioma were found, on pathological examination, to be due to inflammatory exudation into the vitreous, with or without detachment of the retina. An attentive perusal of the details shows that of the eleven cases several were instances of retinitis exudativa. The communication, it should be added, was written sixteen years before George Coats clearly differentiated the condition from a crowd of others (1908). An instructive case of the kind was published recently by a Japanese surgeon, Hajano,⁵

¹ *Trans. Ophth. Soc. U.K.*, 1883, iii, p. 129.

² "Researches into the Anatomy and Pathology of the Eye," 1896.

³ A later and in some respects perhaps a better presentment of the condition was published by George Coats in *v. Graefe's Arch. f. Ophth.*, 1912, lxxxi, pp. 275-329.

⁴ *Roy. Lond. Ophth. Hosp. Reports*, December, 1892, p. 361.

⁵ *v. Graefe's Arch. f. Ophth.*, 1913, lxxxiv, i, p. 30.

where the eye of a small boy, aged 2, was enucleated as an instance of true glioma. On pathological examination, there was found complete detachment of the thickened and folded retina, the deeper layers of which showed changes believed to be those of retinitis exudativa.

The first case that drew my attention to the subject was in a little boy, aged $2\frac{1}{2}$, who was brought to me in May, 1908, because he "had a kind of cast in his left eye." Since an attack of influenza, three months before I saw the child, the eye had been inflamed from time to time, and the father (an observant person) had occasionally noticed a shining appearance "like that from a cat's eye." On examination, the patient's left eye was found to be blind, and its tension was thought to be raised. There was slight ciliary redness. The pupil was quadri-lateral in outline and motionless to light. With the mirror, areas of



FIG. 1.

clear fundus could be made out here and there, interspersed with greyish-white masses of flocculent material. Several groups of strikingly bright spots and patches (difficult to locate exactly) could be seen in various parts of the fundus. A hæmorrhage was present in the lower-outer part of the vitreous. The right eye, it should be added, was normal in all respects. It was thought that the condition might possibly be one of glioma endophytum, and the eye was removed under that supposition. The eyeball, which measured 20 mm. by 20 mm., was frozen, and divided into a nasal and a temporal half by a vertical antero-posterior section. To the naked eye, the anterior part of the globe showed no obvious change, except for a rather striking blunt projection in the neighbourhood of the posterior pole of the crystalline lens, the so-called "moulding" (fig. 1). The retina was nowhere grossly detached.

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The cross-section of a greyish-white nodule (5 mm. by 2 mm.) lay between choroid and retina far forward near the ora serrata, and was evidently part of a larger mass in the same region. Signs of other nodules were to be dimly seen scattered haphazard over the remaining parts of the hemi-section. Several groups of shining points, probably cholesterin, were present here and there.

On pathological examination, nodules of newly formed fibrous tissue were found in the outer layers of the retina, particularly in the vicinity of the upper part of the ora serrata, where larger masses had

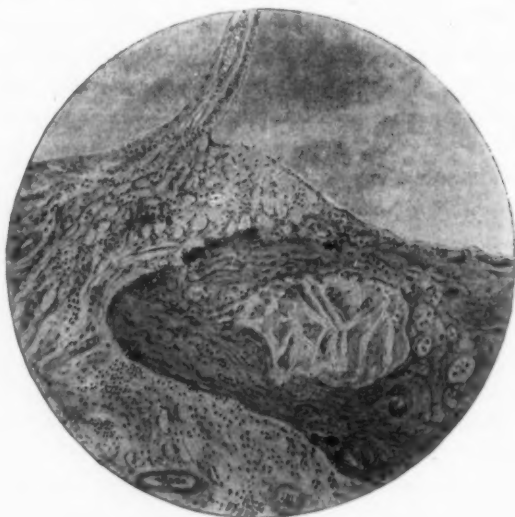


FIG. 2.

evidently been formed by the aggregation of smaller ones. The nodules were permeated by a system of clefts, which doubtless once had held cholesterin crystals, dissolved out by the ether and other chemicals used in making the preparations. They included for the most part a good deal of pigment, and contained larger or smaller collections of giant cells and of disintegrating blood-clot. The retina was degenerate and more or less disorganized, although the outer nuclear layer could be recognized in places. The lumen of many of the retinal vessels was narrowed or displaced by hyaline changes (fig. 2).

In brief, then, a typical nodule lay in the depths of the retina, and

was composed in the main of newly formed fibrous tissue, coloured red by van Gieson's stain, and permeated by an extensive system of cholesterin clefts (fig. 3). It contained numerous pigment particles, and enclosed one or more cavities of irregular shape, which often intercommunicated and were frequently more or less filled with the remains of disintegrating blood in the shape of debris, degenerate blood corpuscles, particles of pigment, networks of fibrin, swollen leucocytes, and giant cells. Neither calcareous nor caseous changes were found. In many of the sections the choroid could be traced unaltered over the



FIG. 3.

nodules, but in some the two structures appeared to be in organic connexion. For the rest, the angle of the anterior chamber was closed. The tissue of the iris was atrophic, with pigment clumps here and there, and it had evidently been inflamed. The subcapsular epithelium extended around the lens.

For the other case I am indebted to Mr. Arthur B. Zorab, of Southampton, to whom the patient, a little girl, aged 4, was brought in February, 1914, because the left eye appeared to be blind and had been noticed to turn inwards. Mr. Zorab found that the tension of the affected eye was +1, and that a bluish-grey reflex could be obtained

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from the fundus with the ophthalmoscope. After dilatation of the pupil, a complete detachment of the retina was observed. The retina could be traced up to its most anterior limits all round, except for a small area to the outer side, where a "dip" could be made out. By focal illumination the retinal vessels could be seen. Mr. Zorab thought it probable that the eye contained a glioma, and it was accordingly excised. The specimen was placed in 1 per cent. formalin and forwarded to me for examination. The child was seen by Mr. Zorab in September last, that is, upwards of eighteen months after the operation, when



FIG. 4.

the general health was found to be good, and the remaining eye showed no signs of any mischief.

The pathological condition of the excised eye was briefly as follows: The retina was detached *in toto*. The subretinal space was filled with an abundant homogeneous coagulum, containing "ghost cells" and cholesterin clefts. Although degenerate, the nuclear layers of the retina could usually be recognized as such. In places the retina was infiltrated with "ghost cells" similar to those present in the subretinal coagulum. In the outer layers of the retina, more particularly in the neighbourhood of the ora serrata, were nodules of two kinds, both of which had

evidently been formed from extravasations of blood. The first took the form of more or less homogeneous collections of fibrin or of newly formed connective tissue (fig. 4). The second occurred as collections of pigmented tissue cells, intermingled with new fibrous tissue and remains of blood corpuscles. These included tolerably numerous cholesterol cracks or clefts. For the most part the choroid was quite distinct from the retinal nodules, but in one small mass of reniform shape the two appeared to be in structural continuity through imperfections in the pigment epithelium (fig. 5). Many of the retinal

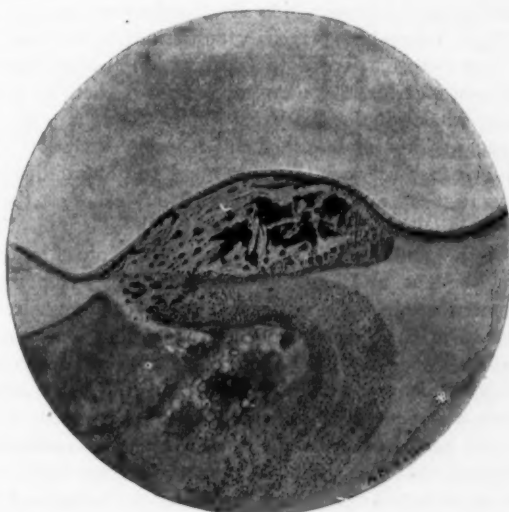


FIG. 5.

vessels were thrombosed; and hyaline changes, leading to narrowing or displacement of the lumen, were not uncommon. The choroid showed no particular lesions. As regards the anterior half of the eyeball, the ciliary body and the iris were atrophic, and the uveal pigment could be seen to extend around the edge of the iris (ectropion uvae). The posterior pole of the lens was somewhat "moulded." The angle of the anterior chamber was closed.

In conclusion, I should like to take this opportunity of expressing my gratitude for the work of the late Mr. George Coats, who examined some of my specimens and confirmed the pathological diagnosis of the foregoing cases.

DISCUSSION.

The PRESIDENT: Are there any forms of pseudo-glioma in which, provided a positive diagnosis can be made, the eye should be left in the orbit? In two instances I have removed an eye which appeared to contain a glioma but in which the opacity was a developmental defect connected with a persistent fetal artery of the lens. Would those eyes have given trouble later? Inflammatory opacities simulating glioma I think generally call for excision.

Mr. TREACHER COLLINS: I have excised one eye, supposing it to contain a glioma belonging to the class which Mr. Stephenson has just described. I presume that these cases had primarily subretinal hæmorrhage and that the fibrous tissue demonstrated is due to organization taking place in the blood-clot. In these cases, as Mr. Stephenson has shown, there is an entire absence of inflammatory exudation in the uveal tract. The late Mr. Coats himself held that the cases which he described as "massive exudation" were probably, primarily, hæmorrhages between the retina and the choroid, where afterwards there was fibrous tissue formation in the clot. These cases are therefore different to the cases of so-called pseudo-glioma, in which there is a plastic inflammatory exudation from the ciliary body, which organizes into fibrous tissue in the vitreous behind the lens, and in which the similarity to glioma is intensified by the development, in that fibrous tissue, of new blood-vessels. I think these latter cases can often be differentiated from true glioma by the fact that the blood-vessels come from the ciliary body, and are therefore biggest at the periphery, branching as they proceed towards the centre. On the other hand, where there is a glioma with the retina detached over it, the blood-vessels are largest in the centre and branched towards the periphery. Another point of differentiation is that the plastic exudation from the ciliary body often passes forward into the circumlental space, attaches itself to the root of the iris, and in organizing retracts it so that the anterior chamber becomes deepened at the periphery. There is also another class of cases of pseudo-glioma, which the President has mentioned, in which there is a congenital development of fibrous tissue behind the lens, often with persistence of the central hyaloid artery. Many such eyes have I seen taken out under the suspicion that they contained glioma. I think that this condition may be differentiated by finding that a red reflex can be obtained through the periphery of the lens in all directions. Usually, the fibrous tissue does not extend quite up to the ciliary process, and the central hyaloid artery can be seen in the centre of the fibrous tissue. As a glioma always comes from one side, there is not a red reflex obtainable in the entire circumference. The class of case which Mr. Stephenson has described this evening is the most difficult to differentiate from glioma, and I should like to hear whether Mr. Stephenson can suggest any points of distinction. In my own case of the same kind there was a history of some birth injury, and I consider that there was a subretinal

hæmorrhage at birth, in which fibrous tissue subsequently developed. In an infant it will easily be understood how such a condition can be mistaken for glioma.

Mr. W. H. H. JESSOP: Four years ago I had a case, in a patient aged 12, with a condition of the eye which exactly resembled glioma. No history could be procured as the child was a foundling. After excision of the eye, it was found to be a developmental case. I should like to know if anyone present has seen a case of glioma over 7 years of age.

Mr. STEPHEN MAYOU: I have had a similar case to that which Mr. Stephenson has already shown, and which I published in the discussion on detachment of the retina before the Ophthalmological Society in 1915. The patient was a boy at Paddington Green Children's Hospital, aged 12, who came with acute glaucoma in one eye. After he had been in hospital two or three days the fundus could be seen; there was a retinal detachment with a hæmorrhage seen on it. The eye was removed, and pathologically it was seen to have almost the identical appearances which Mr. Stephenson has just described. Knowing of the case of the late Mr. Coats, I concluded it was of the same nature. But I took care to cut the *venæ vorticosæ* in series, and found them thrombosed. Whether that was the primary cause of the condition I am not prepared to say. In that case there was a history of injury about six weeks before the onset of the glaucoma.

Mr. A. ZORAB (Southampton): I should like somewhat to amplify the report of the case which I sent to Mr. Stephenson, though I have nothing to say about the sections. There is one point connected with the history which is impressed on my mind by the difference in the two cases which Mr. Stephenson has described. In the first case, the fundus could be carefully examined, and there were different parts recognized in it, nodules and bodies or bits of detachment. I should like to state the history told me by the parents, because the parents were observant people, and when I heard Mr. Stephenson was to read a paper, I looked up my notes. There was a squint a year before I was asked to see the child. The parents did not think seriously about the squint, being under the impression that she would grow out of it. They noticed that the pupil was originally black, but it had been gradually growing greyer, till at length it began to look like a cat's eye. I regarded that as fairly definite evidence that there was something progressive in the eye. I saw nothing of the fundus but the retina up against the back of the lens, but there was one little cleft to the outer side, and had it not been for the presence of the retinal vessels I should have been hard put to it to make a diagnosis of detached retina. When the eye had been removed I sent it to Mr. Stephenson, and when I received the report, I placed the matter before the parents, telling them I had thought it was a malignant eye, and therefore it must be removed, but that it now appeared it was not malignant. The sentiments of the parents seemed to be divided between relief at finding it

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was not malignant, and regret that an eye had been removed which was not likely to be dangerous. But I think I did my duty, because the eye was blind and might have been a source of danger, and an artificial eye looks better. This afternoon I saw another case in an infant, aged 4 months. The mother had noticed the same thing ever since birth—a grey reflex from the right eye. After I had dilated the pupil I saw the condition which Mr. Treacher Collins has just spoken of. The "opacity" was seen to spread only three-fourths of the way across the back of the lens; it was not the ordinary grey opacity seen in lamellar cataract, but the grey shiny appearance of detached retina. There was a distinct red reflex round the outer side of it, and I caught sight of retinal vessels at the side, but, of course, examination of the eye in so young a child was difficult. I was anxious, because, when it stretched up to the ciliary body on one side, the chances were in favour of there being glioma as has just been pointed out by Mr. Collins. I should be glad if any member would tell me whether that eye ought to be removed. There is no squint at present. One can see clearly to the back of the fundus at the outer side of the eye. I have not seen any similar cases other than these two. In answer to Mr. Jessop's question, the oldest case of glioma in the records of Southampton Hospital is—speaking from memory—6 years of age.

Captain ORMOND: Mr. Devereux Marshall published, in the Moorfields Hospital Reports, a case of glioma retinae of 11 years of age. Otherwise I think the oldest recorded case is 8 years of age. The case of the patient, aged 11, was reported by Professor Hirschberg.

Mr. SYDNEY STEPHENSON: I congratulate myself on the fact that my paper has given rise to so interesting a discussion. I wish particularly to thank Mr. Treacher Collins for his practical remarks with regard to the diagnosis of some of these conditions.

(November 3, 1915.)

Late Results of Operative Treatment of High Myopia.

By A. HUGH THOMPSON, M.D.

FIVE years ago I published some notes based on my experience of twenty myopic eyes, the clear lenses of which I had needled, with the object of making the refraction approximately emmetropic.¹ It may be of some interest to put on record my further experience of those of these same cases with which I have been able to keep in touch. I may say that during the last five years, probably in common with most ophthalmic surgeons, I have been decidedly chary in recommending this operation; but I still maintain that in a few carefully selected cases the treatment is a good one, and tends to make the life of those patients who undergo it decidedly more satisfactory than it would otherwise have been. Since, however, we can never speak with anything like confidence of the future of these eyes, my opinion is strong that one should never press the operation on any patient, and we should be on our guard against representing in the light of a cure a procedure which leaves the eye just as likely as before to be attacked by those destructive processes to which, as is well known, highly myopic eyes are subject. On the other hand, it has, in my opinion, yet to be proved that after a successful operation in a well-selected case, the eye is any more subject to those same destructive processes than it would have been if it had been left alone. Meantime the patient, at any rate for a time, enjoys the advantages of greatly improved vision.

Before proceeding to the subject of my paper—the actual condition of those eyes on which I operated more than five years ago—I will say a few words first on the selection of cases, and, secondly, on the particular operations performed.

The degree of myopia which is in my opinion most suitable for operation is anything between 16 and 22D. If we needle eyes whose myopia is less than 16D., we find it necessary to supply the patient with a convex glass afterwards, and the advantage derived by the patient is not so very great. In the case of young children, however, whose

¹ *Ophth. Rev.*, 1910, xxix, pp. 821-835.

myopia is certainly progressive, I think we might put the limit lower, say at 14D. If, on the other hand, the myopia is more than about -22D., the chances of the eye being or remaining free from those destructive processes to which all highly myopic eyes are liable is comparatively small. I do not consider suitable for operation an eye with much choroiditis, and, above all, an eye whose macular region is already obviously affected by degenerative changes. As to age, all my patients have been between the ages of 6 and 25. I have had two little patients, aged 6, whose lenses I have needled.

My method of operating was the usual one—i.e., a free needling of the lens capsule. It is then necessary to watch the patient carefully day by day, and in case of rise of tension to let out the lens matter by a keratome incision through the cornea. Otherwise one can allow the lens matter slowly to absorb, and perform a second needling, if necessary, at a later date. Occasionally a secondary membrane forms some years after the original operation, and it is necessary to needle this. In two of my cases I have found it necessary to divide adhesions between the lens capsule and the keratome scar in the cornea. On an average the number of operations required for each eye operated on from beginning to end has been in my cases between three and four.

The number of patients on whom I had operated before the middle of the year 1910 was nineteen. Of these one, dating from 1899, was a failure from the first, owing to cyclitis and a thick cyclitic membrane. It was not a case that in more recent years I should have considered a suitable one for operation at all. A second case was complicated by congenital dislocation of both lenses, and as the case was not a typical one, I am omitting it for the purposes of this paper. I may say, however, that the result of operation in this patient's case was entirely satisfactory, and equally so in her second eye, on which I operated two years ago. Besides these two cases there were three in which the results of operation were satisfactory, so far as I was able to follow them up; but as I lost sight of them before the expiration of four years, I am not including them in this paper. This leaves fourteen patients whom I have been able to follow up during periods of from five to fifteen years. Of these fourteen there was one in whom I operated on both eyes, but I may say that I consider the advantage to be gained by operating on the second eye so comparatively slight that it is not in most cases worth taking the risk. In that one case I yielded to the importunity of the patient, but I should not be inclined to do so in a similar case again, though there was no untoward result in her case.

There are, then, fifteen eyes, in all of which the immediate results of operation were good, which I now propose to consider from the point of view of their condition from five to fifteen years after the first operation. I have set down a summary of my results in tabular form (*see* Tables I and II):—

TABLE I.

Name	Sex	Age at time of first operation	Diopters of myopia	Best vision with a glass before operation	Number of years from first operation, with latest date seen	Best vision without glass at latest testing	Best vision with glass at latest testing	Remarks
A. L.	F.	17	-22	$\frac{5}{80}$	6 to 1911	$\frac{5}{80}$	$\frac{1}{8}$	—
R. E.	F.	17	-17	$\frac{1}{15}$	9 to 1913	$\frac{1}{15}$	$\frac{1}{15}$	—
R. A.	F.	25	-16.5	$\frac{1}{32}$	10 to 1914	$\frac{1}{11}$	$\frac{1}{15}$	(1914) macular changes in unoperated eye; hæmoptysis, aged 35
R. B.	F.	16	-16.5 (left eye)	$\frac{1}{15}$ (2 let.)	8 to 1912	$\frac{1}{15}$	$\frac{1}{15}$	—
		19	-14.5 (right eye)	$\frac{1}{15}$ (2 let.)	5 to 1912	$\frac{1}{14}$	$\frac{1}{15}$	—
I. C.	F.	9	-16	$\frac{1}{32}$	8 to 1914	$\frac{1}{15}$ (2 let.)	$\frac{1}{11}$	Mother of the patient a high myope with macular degeneration
L. F.	F.	6	-16	Too young to test	7 to 1915	$\frac{1}{15}$	$\frac{1}{15}$	—
S. P.	F.	17	-19	$\frac{1}{15}$ (2 let.)	7 to 1915	$\frac{1}{15}$	$\frac{1}{11}$	—
W. W.	M.	22	-20	$\frac{1}{80}$	5 to 1915	$\frac{1}{15}$	$\frac{1}{15}$	—
M. T.	F.	22	-16	$\frac{1}{15}$	8 to 1915	$\frac{1}{15}$ (2 let.)	$\frac{1}{15}$	Macular hæmorrhage eight months after operation; this cleared up, leaving no trace

The great preponderance of females to males will be noted in this series of cases. This I attribute partly to accidental causes, and partly to a real preponderance of high myopia in the female sex.

Now with regard to the condition of these eyes several years after the lens had been removed. It will be seen in Table I that ten eyes—i.e., two-thirds of those operated on—have after an average period of eight years materially better vision than they had before operation, and that nine out of ten of these eyes obtained vision of $\frac{6}{24}$, $\frac{6}{18}$, or $\frac{6}{12}$ without wearing any glass at all. It is to be noted that in one of these successful cases (R. A.) macular changes have occurred in the unoperated, but not

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in the operated eye. In another (M. T.) a macular hæmorrhage occurred eight months after operation in the operated eye, but this cleared up completely, leaving good vision ($\frac{6}{12}$) eight years after operation. If we now turn to Table II, we find that in five cases—i.e., in one-third of those operated upon—the eyes have gone more or less wrong after enjoying good sight for varying periods up to ten years. In three out of the five the cause was macular degeneration, such as is apt to occur in highly myopic eyes, whether operated upon or not. In two of the three (L. B. and A. B.) the macular degeneration was binocular—i.e., it occurred in the unoperated and operated eyes alike. In the third case (D. H.)

TABLE II.

Name	Sex	Age at time of first operation	Diopters of myopia	Best vision with a glass before operation	Number of years from first operation, with latest date seen	Best vision after operation	Duration	Latest test of vision	Cause of bad vision and remarks
L. B.	F.	6	-18	$\frac{6}{20}$	15 to 1915	$\frac{6}{18}$	10 years	$\frac{6}{36}$	Macular hæmorrhage and degeneration in both eyes
M. M.	F.	12	-16	$\frac{3}{8}$	11 to 1915	$\frac{1}{8}$	6 years	$\frac{6}{36}$	Choroiditis; an attack of cyclitis six years after operation, which cleared up
D. H.	F.	15	-17.5	$\frac{6}{36}$	8 to 1913	$\frac{4}{18}$	Uncertain	$\frac{6}{36}$	Macular degeneration
A. B.	M.	18	-20	$\frac{6}{18}$ (11et.)	7 to 1914	$\frac{6}{18}$	5 years	P. L.	Macular degeneration; second eye also affected with macular degeneration, but vision -21 = $\frac{6}{18}$.
R. J.	F.	21	-18	$\frac{6}{18}$	5 to 1913	$\frac{6}{18}$	4 years	$\frac{6}{36}$	Iritic membrane; pyorrhœa alveolaris

the unoperated eye was only myopic to a comparatively small extent (-5D.), so that it was not equally liable with the operated eye to this disaster. Further experience therefore confirms the conclusion that I came to five years ago that the most common disaster which highly myopic eyes are liable to is macular disease—degenerative, inflammatory, or hæmorrhagic—and that the fact that they have or have not been operated on makes no difference to this liability. The remaining two cases, now enjoying but poor vision, are of some interest. R. J., the last case in Table II, for some years after the first operation enjoyed fairly good vision, so that she was able to do her work as a domestic

servant without glasses. Four and a half years after the first operation she came again to the hospital with synechiæ binding the pupil all round to the remains of the lens capsule and a thin membrane covering the pupil. This membrane was easily needled, but every time I needled it it formed again, so that the latest vision of the eye was only $\frac{6}{60}$. There was one circumstance about this case that made me think that it was one on which I may have operated too hastily, and that was that the patient was the subject of pyorrhœa alveolaris, and though I sent her to the dentist to have the mouth put into good order before operation, I am afraid that this was not done with sufficient thoroughness.

The other case was that of M. M., who, after enjoying $\frac{6}{18}$ vision without a glass ($\frac{6}{12}$ with a glass) for six years, came back to the hospital with a thick crop of keratitis punctata and a defective field. I suspected a detached retina at the time, but this proved not to be so, for the keratitis punctata cleared up and the fundus showed no detachment, but a large patch of choroiditis together with macular changes, which explained both the defective field and the bad central vision ($\frac{6}{60}$). Whether or not there was any connexion in this case between the uveitis and the removal of the lens I am not prepared to say.

It will be noted that in not one of this series of cases have I had to deal with detachment of the retina as a sequela to the operation for high myopia. That it does occasionally occur, however, is well known, though whether a highly myopic eye is more subject to it after operation than before is doubtful. My own feeling is that in eyes with healthy fundi this is not so, but that where a tendency to detachment already exists it may very well be confirmed by operative interference. In this connexion I will relate a case which came under my care at a late stage.

L. K., female, aged 25, with myopia of $-21D$. in the right eye and $-25D$. in the left, and some central choroiditis in the left, had her left lens needled by my colleague, Mr. Inglis Taylor, in the year 1907. Till 1913 the vision of that eye without a glass varied from $\frac{6}{9}$ to $\frac{6}{12}$, but soon after the operation a detachment of the retina took place in the right (unoperated) eye. In 1913, six years after the last operation, the sight of the left (the operated) eye began to fail, and a detachment was noted in that eye, too. Meantime, however, the detachment in the right eye had subsided, so that it could no longer be diagnosed with the ophthalmoscope. In the forlorn hope of restoring to the patient some useful vision the right lens was now needled and lens matter evacuated. The detachment, however, was still present, and no useful vision was obtained.

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I think that this case is very instructive. It shows: (1) That an eye with myopia of as much as $-25D$. and central choroiditis may be needled with temporary success; (2) that such an eye, if it is not attacked by destructive changes sooner, is very liable to succumb to them later; (3) that if detachment of the retina occurs in such an eye after operation it is not necessarily due to the operation, but would very likely have occurred if no operation had been performed; and (4) to remove a lens from an eye in which detachment has already taken place is only to court disaster.

TABLE III.—GROWTH OF LONG AXIS OF EYEBALL.

Initials	Refraction of operated eye	Rate per year in diopters	Refraction of second eye	Rate per year in diopters
L. B.	After operation, $+5.0$ 14 years later, emmetropic	0.36	Time of operation -18.5 14 years later -32.0	0.96
A. L.	After operation -2.0 5 years later -3.5	0.30	Before operation -19.5 6 years later -20.5	0.17
R. E.	After operation $+1.75$ 9 years later, emmetropic	0.19	Before operation -17.0 9 years later -22.0	0.56
M. M.	After operation $+3.0$ 11 years later $+1.0$	0.18	Before operation -15.0 11 years later -22.0	0.64
R. A.	After operation $+1.25$ 10 years later $+1.0$	0.22	Before operation -13.5 10 years later -18.0	0.45
M. T.	After operation $+1.5$ 8 years later $+1.0$	0.06	After operation -9.0 8 years later -11.0	0.25
A. B.	After operation $+3.0$ 7 years later -1.5	0.64	Time of operation -15.0 7 years later -25.0	1.43
I. C.	After operation $+3.0$ 7 years later, emmetropic	0.43	Before operation -15.0 7 years later -24.0	1.29
S. P.	After operation $+2.5$ 7 years later $+0.5$	0.29	Before operation -15.0 7 years later -16.5	0.21
L. F.	After operation $+4.0$ 6 years later $+1.0$	0.50	Time of operation -13.0 6 years later -17.0	0.67
D. H.	After operation $+1.5$ 8 years later, emmetropic	0.19	Before operation -4.5 8 years later -7.0	0.31
	Average rate per year ...	0.3	—	0.63

The question is sometimes asked whether the operative treatment of high myopia tends to counteract the tendency of the long axis of the eyeball to increase. I do not think that it does tend to counteract it.

On the other hand, the fact that the lens has been removed does, for obvious optical reasons, diminish the effect of the lengthening of the axis of the eyeball on the refraction by just about one-half. In those patients whom I have been able to follow up for a number of years I find, by comparing the refraction of the operated with that of the unoperated eye, results roughly corresponding to what one would expect on the assumption that the operation in itself has no effect one way or the other on the growth of the long axis of the eyeball. These results I have tabulated (Table III). Eleven patients were observed for an average period of eight and a half years, with reference to the alteration in the refraction of both their operated and their unoperated eyes. The average rate of growth in the macular refraction in the case of the operated was 0.3D. per year. In the case of the unoperated eyes it was 0.63D. per year—i.e., just double—as one would expect on the assumption that the growth in the long axis of the two eyes tends to be equal. Although I do not pretend that my retinoscopy in all these cases of high myopia was accurate to half a diopter, I think it will be admitted that the average obtained from these eleven cases is sufficiently striking to prove my point.

DISCUSSION.

Mr. HARRISON BUTLER (Leamington): When I was in Jerusalem some years ago, I did two of the operations mentioned in the paper, one on an Arab boy and the other on an Arab girl; the result was good in each case, $\frac{2}{3}$ acuity being attained with +1 and -1 respectively, but after a year, for no apparent reason, the retina became detached in one of the cases. Therefore, since that date I have felt chary of doing the operation.

Mr. R. COULTER (Newport, Mon.): During one period, when I was assistant to Mr. Cross, I saw a good deal of cases such as those described in the paper, and I have subsequently needled two lenses. As both eyes went wrong, I abandoned the procedure. I have had under observation for several years one patient from whom the lens was removed by another surgeon. The result was very good at first, but the eye went wrong subsequently from macular damage. Since I have known the patient he has had two or three attacks of retinal hæmorrhage in the unoperated eye, which have now cleared up, leaving useful vision.

Mr. ARNOLD LAWSON: I consider that the operation dealt with in the paper should be viewed with a good deal of caution; but it is a procedure which will not be dropped if the cases for it are carefully selected. When the operation first came into vogue, the cases were not properly selected, so that

there was a tendency to undertake operations when they should not have been performed. Several points in Mr. Thompson's paper are of interest to me. The first is as to the amount of myopia. The author says that his cases have mostly between 16D. and 22D. of myopia, and within those limits Mr. Thompson found no glasses were required after the operation. My own experience is that one cannot expect to secure a good result without convex glasses unless the myopia amounts to -20D. I have not seen a case of 20D. without some choroidal change; and that being so, it should be recognized that all these highly myopic eyes are prone to degeneration from only slight causes. Therefore, if the surgeon decides that the degeneration seen in such an eye is not too bad to make him say he should not do the operation, he should be very careful how he does the needling. Upon that point I wish to lay stress. I do not think it should be a free needling, but a very light needling; and the great object in view should be to avoid a curette evacuation, because that is the great danger. After a curette evacuation, it is very common to find chronic inflammation set up; and in these highly myopic eyes, which are so prone to degenerative changes, damage may be produced which will tend to the eventual loss of the eye. In addition, curette evacuation favours detachment, because it must be in the experience of all members of the Section that in not a few such cases one sees the vitreous coming forward through a too free rush of lens debris through the wound. In my view, it is detachment of the retina which one has most to fear after needling for high myopia. I cannot say I have ever seen more degenerative changes take place after needling these cases of myopia, and I do not admit that the risk of detachment is great after needling these cases if the cases for the operation are properly selected and suitably treated. I have had several excellent results, and some of my patients who were operated on in this way ten and eleven years ago are still doing well. I am strongly of opinion that operative interference should be confined to one eye.

Mr. W. H. H. JESSOP: I agree with the remarks of Mr. Lawson in opposition to doing a curette evacuation in these cases. Years ago, when the procedure first came in, I had many such cases, and the first was that of a boy who had 24D. of myopia, and with correction he had vision $\frac{5}{16}$. I operated first on the right eye, when the vision, without glasses, became $\frac{9}{16}$, a result I could scarcely understand. It being my first case, encouraged by the result, I did the same in the second eye, and with this also the boy saw $\frac{9}{16}$ without glasses. But since then I have not had such a good result. I seldom do the operation now because I, too, fear the occurrence of detachment, and it will be interesting to hear how Mr. Thompson has managed to escape detachment occurring in his cases. The boy I have referred to went on for six years, and then had sudden detachment in one eye, but the other has preserved normal vision. I have not done the operation in both eyes of the same patient since.

Mr. W. T. HOLMES SPICER: My first case was on much the same lines as Mr. Jessop's first. The patient was a girl with 24D. in one eye and 26D. in the other, and vision $\frac{5}{16}$ in each. It occurred sixteen years ago. After the

needling there was $\frac{1}{12}$ vision in one eye, $\frac{9}{16}$ in the other without glasses. I desire to support Mr. Lawson's remarks. My own experience is that the great drawback in these operations is not so much the risk of detachment of retina as of the occurrence of macular hæmorrhage, followed by macular degeneration. The only cases in which I have seen detachment of retina have been those after a curette evacuation, in which an anterior synechia has been left. One was the case of a girl, on one eye of whom Mr. Nettleship operated, and who came under my care with the second eye. A curette evacuation of the lens had been done, and in both eyes there was an anterior synechia, the iris being caught up to the evacuation wound. She subsequently had a detachment of the retina in each eye. Nowadays such a synechia would be divided, and the danger of detachment diminished. The danger of detachment of retina with an anterior synechia is as great in non-myopic eyes—for example, lamellar cataracts.

The PRESIDENT: I think Mr. Thompson's tables will be of permanent value. My own experience of such treatment has been small and not encouraging. It will be instructive to learn, not only the ultimate fate of eyes that recover well from the operation, but to know what proportion of such cases immediately go wrong.

Mr. ZORAB (Southampton): I have been performing the operation fairly freely during the last four years, but I have never had in view the object of producing an emmetropic eye. I would have liked to hear what has guided other members in deciding to advise the operation. I have set before myself two standards: (1) If the corrected vision is insufficient for the patient's purposes and there is no other contra-indication, I am prepared to advise it; (2) if corrected vision is good enough for the patient's purposes, but the wearing of such powerful lenses is impracticable or contrary to local regulations, I advise operation.

Mr. A. HUGH THOMPSON (in reply): I am rather fortunate in this operation, because I have had only one calamity certainly due to the operation. Up to the year 1910, however, I had only done the operation on twenty eyes, and fifteen of these were included in the paper. Since that year I have done it only three times, and two of them have turned out perfectly well. The last I did is in course of treatment now; I had to do a curette evacuation, and I am anxious about it. In the other paper which I wrote, I made some calculations from all my cases, and I found that the average formula was half the myopia previous to the needling plus 10'5, so that a myopia of 20 will leave a hypermetropia + $\frac{1}{2}$, practically what Mr. Lawson has said. It is not necessary to get exact emmetropia after needling in order to enable a patient to see fairly well without a glass. Most patients get $\frac{1}{8}$ or $\frac{1}{12}$ without a glass, one or two lines better with a low plus glass. I have also been very lucky with regard to detachments in my cases, because in not one of the eyes I have operated upon has detachment occurred. There was one case in which, when I wrote my former

paper, I thought there had been detachment; it was that in which I mentioned keratitis punctata was coming on suddenly six years after the operation and a defective field. That keratitis punctata cleared up, and there was no detachment, only a large patch of choroiditis. I know that detachments do occur. [The PRESIDENT: If you correct the myopic eye you get the retinal picture of a certain size, and if you take the lens away you will again have good definition but a much larger retinal image, which will account for the occasional result of $\frac{6}{6}$.] I think that is so, Mr. President. In reply to Mr. Zorab, I would say one must consult the patients or, if the patient be a child, its parents. Generally the patients are between 16 and 22 years of age, and then it is a question of carrying out their occupation. All the cases dealt with in my paper are hospital patients, who have to earn their living, and the operation makes a good deal of difference to their capacity in that respect.

Section of Ophthalmology.

President—Mr. PRIESTLEY SMITH, F.R.C.S.

(February 2, 1916.)

Case of Aneurysm of the Cavernous Sinus.

By J. HERBERT FISHER, F.R.C.S.

In presenting this case of antero-venous aneurysm of the cavernous sinus, I would like to take the opportunity of referring to a case of the same character which I showed before this Section of the Society in May, 1913. I was able to watch that case until October, 1913, but have not been successful in tracing the patient subsequently. At that date (October, 1913) the vision of the left eye was $\frac{6}{60}$. There was still some defect in the power of elevation of the globe, and slight ptosis remained, although all other movements were normal. The pupil was wide. There were still a few large veins on the surface of the sclerotic, near to the cornea. Ophthalmoscopic examination revealed no departure from the normal, but the eye showed the appearances of a congenital crescent on the nasal and lower edge of the disk, and it was not certain that there may not have been some congenital amblyopia in this eye. In this case, at any rate for ten months after the common carotid artery was ligatured, no recurrence of pulsation had taken place.

The present patient is Mrs. E. C., aged 59, whom I saw at St. Thomas's Hospital on October 21, 1915. Seven weeks earlier she had bolted her front door and ascended about six stairs on her way to bed when she suddenly felt giddy and fell to the bottom of the staircase. In her opinion she did not in any way slip or trip on the stairway. She remained unconscious for four or five hours, and vomited on recovering

her senses, and was sick on occasions during the subsequent two or three days. She complained of pain mainly in the right forehead. On the third day, according to her doctor's statement, the right eye became prominent; the lid had drooped ever since the accident. When I examined her there was complete ptosis, some puffiness of lids, distension of veins in the subcutaneous tissue of the upper lid, complete ophthalmoplegia externa with the exception of such movements as could be effected by the superior oblique muscle. The pupil was fixed, and gave no reaction to light; the vision $\frac{6}{60}$, and, with allowance for 2D. of hypermetropia, $\frac{6}{24}$ partly. There was marked pulsation of the right eyeball; this was easily felt and seen, and a thrill is readily felt when palpation is made on the inner half of the upper lid. A loud buzzing bruit was audible over the eyeball and the right fronto-temporal region, but the patient complained of no noise in the head. Ophthalmoscopic examination showed that the right retinal veins were probably somewhat fuller than those of the left, but not grossly so. No hæmorrhages could be seen. The cutaneous area of the fifth nerve was not insensitive, and the patient appeared to experience very little discomfort from her condition.

I made her an in-patient, which she remained from October 25, 1915, to November 28, 1915. She was kept completely at rest in bed, and 10-gr. doses of iodide of potassium were administered. During this time movements of the eye improved, and also its sight. About December 20 the vision was $\frac{6}{18}$ unaided, and with a + 2.25D. sph., $\frac{6}{6}$. Ophthalmoscopic examination showed slight pumping of the veins in the right retina, more noticeable than in the left, but there was no venous nor arterial pulse contraction in either eye. The right pupil is now less than the left in a not very strong illumination. When cocaine is instilled into the eyes the left pupil dilates in the usual way, but the right gives little or no response. There is, therefore, some evidence to support the view that the dilating fibres of the right iris are impaired in their action, this being due to implication of the sympathetic nerve filaments in the cavernous sinus. In the last month no marked change has taken place.

It seems quite possible, therefore, that in this case the communication between the internal carotid artery and the cavernous sinus took place spontaneously, and that the artery was not lacerated by any fracture of the bones of the base of the skull; that the insensibility and fall resulted from the disorder of the cerebral circulation. It is not possible to assign any reason for such a misfortune taking place

spontaneously, but there was certainly no conclusive evidence of a fractured base. The Wassermann test was negative. In this case the subjective discomforts were small, and still remain so; the evidences in favour of a fractured base are by no means conspicuous. No hæmorrhages took place at the time, nor followed the accident, from the nose or mouth, nor was blood found in the vomit.

(February 2, 1916.)

Large Coloboma of the Upper Lid ; Operation ; Result.

By ARNOLD LAWSON, F.R.C.S.

THE accompanying drawings are of a male child, aged 8 months, with a very large coloboma affecting the left upper lid. The inner segment of the lid is much smaller than the outer, and contains only a fragmentary tarsal plate limited to the immediate area about the lid border. There are no lashes on this segment. The outer larger segment of the lid is normal, with the tarsal plate ending abruptly at its inner border. The apex of the coloboma reaches to the upper limit of the tarsal plate. The levator palpebræ tendon, though with only the usual tarsal insertion over about the outer half of the lid, is active over the whole length of the lid, and when the eye is open tucks the coloboma away in the curious manner shown in one of the drawings (fig. 1). Apparently there is some defect in the action of the superior rectus as, when the child is asleep, the cornea fits into the coloboma gap like a picture into a frame, producing a most weird effect (*vide* drawing, fig. 2).

The closure of this large gap was effected more easily than would appear from the drawings. The absence of the tarsal plate on the inner segment allowed a considerable pull from this side. The chief difficulty was the accurate approximation of the opposing lid margins and the formation of the neatest possible scar. These troubles were overcome, after the usual paring of the edges of the coloboma, by using two sets of sutures. With the first set as good an approximation as one could then obtain was effected, and the second set were then inserted to improve matters, whilst the parts were held more or less in position by



FIG. 1.

Coloboma of the upper lid. Drawing showing the appearance when the child was awake and the eye open.



FIG. 2.

Coloboma of the upper lid. Drawing made when the child was asleep. It shows very well the size and shape of the coloboma and the curious framing of the cornea by the gap.

the first set. These were cut out as the need for them disappeared. The first set also showed where special trimming was needed, which could then be done to a nicety.

The only subsequent difficulty was the maintenance of rest in the parts during healing. The lids were fastened together with a couple of sutures, one on each side of the wound, and *both* eyes kept bandaged. On the third day there were some redness and irritation, and it looked as if the sutures were loosening and slightly infected. Warm carbolic



FIG. 3.

Coloboma of the upper lid. Drawing of the lid taken six months after operation when the baby was asleep. There is no notching of the tarsal border and the scar is almost invisible.

fomentations (1 in 80) soon put matters right, and the result obtained is shown in the third drawing (fig. 3). There is no notching of the tarsal border, and the scar is almost invisible.

(February 2, 1916.)

On [the Cause of the Ophthalmoscopic Appearances in
Amaurotic Family Idiocy.

By THE LATE GEORGE COATS, F.R.C.S.

(Read by Mr. R. AFFLECK GREEVES.)

[Mr. R. AFFLECK GREEVES: Before beginning to read this paper I should like to say that I am reading it before this Section in accordance with the wish which Mr. Coats expressed to me just before he went to Edinburgh to undergo the operation which proved fatal.]

In a recent paper,¹ Batten and Mayou record a pathological examination of the condition first described by one of these authors under the name of "Family Cerebral Degeneration with Macular Change." Speaking of the nature of the retinal lesions, the following statements are made: "There is a close relationship between these cases and the cases of amaurotic family idiocy. . . . The only difference in the two diseases appears to be that the macular region in amaurotic family idiocy shows a much more marked oedema of the internuclear layers, and therefore a white area around the macula, which is associated with a hole or thinning of the retina at the fovea due to oedema." This view is reinforced by an account of the histological changes observed in a case of amaurotic family idiocy, in the course of which it is stated that "around the macula there was a very marked oedema of the internuclear layers, which probably corresponds to the white area which is seen during life"; and the conclusion arrived at is that "the oedema of the internuclear layers is present in both diseases, but is very much more marked in the case of amaurotic family idiocy."

It is the purpose of the present communication to dissent emphatically from this description of the histology of the retina in amaurotic family idiocy.

A histological study of the retina, to possess any value, must satisfy three conditions: (1) The material must be absolutely fresh—according to Birch-Hirschfeld, post-mortem changes begin within two hours of

¹ *Proc. Roy. Soc. Med.*, 1915, viii (Ophth. Sect.), p. 70.

death; (2) fixation must be perfect, Zenker's solution being by far the best for this purpose, especially in the intact globe; (3) the method of cutting must be suitable, paraffin being greatly inferior to celloidin, even with the greatest care and with the best fixed material. Insistence on these pre-requisites eliminates nearly everything that has been written on the histology of the retina in amaurotic family idiocy. In my opinion it eliminates the case on which Batten and Mayou rely. It is true that the eyes were injected immediately after death and fixed within an hour; but formalin, the worst of all retinal fixatives, was used, and to judge by the photomicrographs (fig. 5 of their paper), the sections were cut in paraffin.

It would seem that not more than three or four cases have been recorded to which no exception can be taken on the above-mentioned grounds—namely: (1) Cohen and Dixon,¹ eyes obtained in two and a half hours, one fixed in formalin, the other in Orth's solution; (2) Verhoeff,² eyes obtained within fifteen minutes of death, one fixed in formalin, the other in Zenker; (3) Hancock and Coats,³ eyes obtained immediately, within ten minutes of death—one fixed in Müller, the other in Zenker; (4) perhaps also the case of Holden,⁴ eyes obtained four hours after death, one fixed in formalin, the other in Müller. The testimony of these cases is unanimous and unequivocal; in each the changes were absolutely confined to the ganglion cell and nerve-fibre layers, and in each it is specifically stated that there was no oedema, nor was there any folding, thinning, or formation of holes at the fovea. An examination of the fovea in the case described by Hancock and Coats (fig. 1), and a comparison of it with the normal Zenker-fixed fovea (fig. 2), will sufficiently establish the validity of these statements. In fig. 1 the condition of the cones proves that fixation leaves little to be desired; let this photograph be compared with Batten and Mayou's fig. 5. Manifestly, even a single case of this kind would greatly outweigh any number of observations on material less perfectly fixed. Nor is it likely that the difference can be explained by the stage of the process at which the material is obtained, oedema being present at one time, not at another; for it is probable that in all cases the material becomes available at approximately the same point in the histological process—that which corresponds with the death of the patient.

¹ *Journ. Amer. Med. Assoc.*, 1907, xlviii, p. 475.

² *Arch. of Ophth.*, 1909, xxxviii, p. 107.

³ *Brain*, 1911, xxxiii, pp. 514.

⁴ *Journ. Nerv. and Ment. Dis.*, 1898, xxv, pp. 538, 550.

On the question of the similarity in the two diseases of the ganglion cell changes in the retina and central nervous system, I am not competent to pass an opinion. The statement of Dr. Batten that "on the pathological side, the cases were an exact counterpart of each other" must carry the greatest weight. As regards the retina, however, it must be pointed out that, apart from the case of oedema, the other changes of the macula show very considerable differences, which should not be slurred over. Briefly, the changes in family cerebral degeneration are as follows: (1) Certain cytological degenerations of the ganglion cells; (2) a diminution amounting in places to a complete disappearance of these elements; (3) oedema of the internuclear layer; (4) loss of the rods and cones and of the "inner" (surely this should read "outer"?) nuclear layer; (5) thickening of the supporting elements; (6) immigration of pigment into the outer layers of the retina.

Of these only the first two are present in amaurotic family idiocy, and with regard to the second it was a very striking point in the case examined by Hancock and myself, that the diminution in the number of ganglion cells was much less than might have been expected from the degree of degeneration, and nowhere even approximately amounted to a total suppression of these elements.¹

Again, although some hold an opposite view, it is the general opinion of ocular pathologists, founded on the conditions observed in choroido-retinitis and retinitis pigmentosa, that immigration of pigment into the retina of necessity implies a lesion, vascular or other, of the choroid, or at least the action of some noxious agent from the side of the uvea; and the same holds good for degeneration of the rods and cones and outer nuclear layers. But whether or not this rule be of universal application, it is certainly very difficult to believe that a disease which progresses so far as to cause death without producing the least change in the outer layers of the retina, can be the same as one which is evidently associated, even in an early stage, with such changes, as is shown by the disturbance of pigment at the macula. There is, I believe, no known instance of a disease which first attacks the inner retinal neural elements, and then the outer, although instances of the opposite sequence of events might be cited; nor does destruction of the inner layers from whatever cause produce any consecutive secondary change in the outer, however long the duration of the lesion, as is

¹ If the histology of the ganglion cell changes is the same in the two diseases, perhaps this suppression of these cells in family cerebral degeneration accounts for the absence of the typical retinal opacity in that affection.

exemplified, for instance, in the case of obstruction of the central artery or primary optic atrophy, in which the rods and cones and outer nuclear layer remain permanently intact.



FIG. 1.

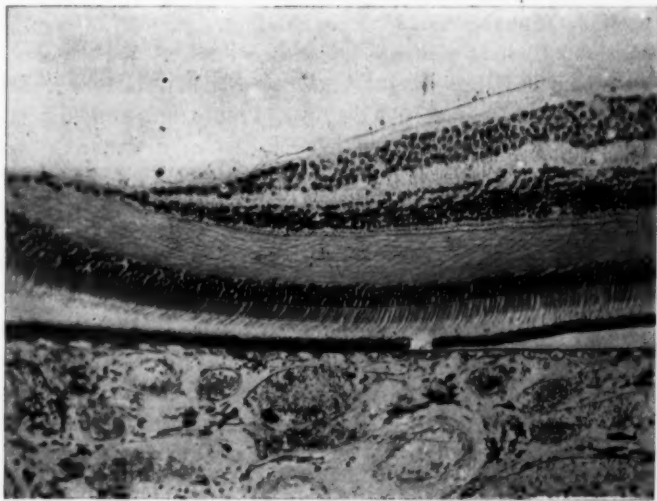


FIG. 2.

These arguments tend to support the view that in spite of a close similarity, or identity of the changes in the ganglion cells, the fundamental pathology of the two conditions is different. Is histological

identity an irrefragable proof of ætiological identity? After all, the tissues of the body have only a limited number of ways in which they can react to noxious influences; amyloid degeneration has the same histological characters, whether it be due to syphilis or tubercle, or to neither of these affections, as in the conjunctiva; fatty degeneration of the liver is much the same whether its cause be phosphorus poisoning, or acute yellow atrophy. Is it not possible, then, that the particular forms of chromolysis, vacuolization, &c., which characterize family cerebral degeneration are closely similar to, or identical with, those of amaurotic family idiocy, while yet the underlying cause is different? The hitherto practically unchallenged race-selectiveness of amaurotic family idiocy should in itself make us pause before we identify that disease with any other which is not race-selective.

I may perhaps be permitted in a brief addendum to repudiate an opinion which was ascribed to me in the discussion on Batten and Mayou's case, but which I have never held, namely, that the ophthalmoscopic appearances in amaurotic family idiocy are due to coagulation necrosis. As its name implies, coagulation necrosis is a change in the tissue proteids probably allied to coagulation, and due to their sudden death *en masse*. Suddenness is of the essence of the process, for if death be protracted, not coagulation necrosis, but fatty degeneration, and other processes, ensue. The dead tissues subsequently undergo various forms of regressive change, invasion, absorption, &c. To attribute to such a process a condition the outstanding features of which are chronicity and persistence over a period of years would be a patent absurdity.

The misunderstanding has probably arisen from the fact that, following Hancock, I have used the case of amaurotic family idiocy in illustration of another thesis originally postulated by Elschnig, namely, that the similar ophthalmoscopic appearances in obstruction of the central artery of the retina, which have also been ascribed to œdema, are due to coagulation necrosis. The argument might be stated as follows: Amaurotic family idiocy furnishes an example of a condition which, although demonstrably confined to the inner layers of the retina, without œdema of the outer, yet produces a white area in the macula and a cherry spot; there is nothing improbable, therefore, in similar ophthalmoscopic appearances being due, in the case of obstruction of the central artery also, to a condition confined to the inner layers of the retina, without œdema of the outer. Not the same condition, of course; in the first case it is a chronic degeneration of the cytoplasm which is

more or less permanent, in the second an acute coagulation necrosis due to cutting off of the blood supply, and disappearing when the necrosed tissues become absorbed.

The point was only a minor link in a chain of evidences supporting the conclusion in question, and was used as an illustration rather than as an essential element in the proof. The other links may be summarized as follows:—

(1) Nowhere in the body does obstruction of an end artery cause œdema. To put it syllogistically: Obstruction of an end artery is followed by coagulation necrosis (white infarction); the central artery of the retina is an end artery; therefore obstruction of the central artery is followed by coagulation necrosis. The retinal changes are in fact a white infarct of the inner layers, which receive their supply from the central artery.

(2) The ophthalmoscopic appearances produced by macular œdema are well known and may be studied in cases of nephritis, papilloœdema, and perhaps Berlin's contusion opacity. They differ considerably from those of obstruction of the central artery; in particular the opacity is never nearly so dense, and there is no cherry spot.

(3) The distribution of the opacity is at variance with the hypothesis that it results from œdema. If due to that cause the opacity should be present in the periphery as well as in the macula, although, perhaps allowing for the greater thickness of the retina at the posterior pole, it might be less in degree in the former situation. There is no reason also why œdema should spare the fovea, and in fact, in the conditions mentioned under (2), it does not do so; pathologically, as is well known, the fovea is a favourite seat of œdema. On the other hand, coagulation necrosis of the ganglion cell and nerve-fibre layers renders a completely satisfactory account of the greater density of the opacity at the macula where these layers are thickest, and of the immunity of the fovea and periphery where they are absent or too thin to be the cause of visible ophthalmoscopic changes.

(4) Edema offers no explanation of the fact that, roughly speaking, the opacity always persists for about the same time. Evidently this time is the period required for the breaking down and absorption of the necrotic layers, not for the absorption of œdema, for it is certain that the rapidity and completeness of the restoration of the circulation is subject to very large variations.

(5) The evidence derived from actual pathological observation is incomplete, the opportunities of acquiring perfectly fresh material from

recent cases of obstruction being of necessity extremely rare. No case appears to have been reported which has been altogether satisfactory from this point of view, and also from the standpoint of perfect fixation.¹ Such as it is, however, the testimony of these cases is equivocal; the changes described as œdematous have been very slight, the formation of small spaces in the nerve-fibre layers, &c., and are manifestly insufficient to account for the extremely dense opacity which is characteristic of the ophthalmoscopic picture. For a discussion of these points, and of the experimental evidence, which is no more conclusive, reference may be made to a paper in the *Royal London Ophthalmic Hospital Reports*, 1913, xix, p. 63.

DISCUSSION.

MR. STEPHEN MAYOU: I feel greatly indebted to the late Mr. Coats for this paper. There are one or two things I would like to say about the paper by Dr. Batten and myself which this contribution criticizes. I am sorry Dr. F. E. Batten is not present. I have not had the privilege of reading Mr. Coats's paper beforehand. When we read our paper I showed two specimens. The first was a specimen from a case of amaurotic family idiocy, and we showed a case of the same type in an older person, which is known as "cerebral degeneration with macular changes." I had the specimen of the amaurotic family idiocy many years ago, and I did not know of any other sections with which I could compare the specimen of "cerebral degeneration with macular changes." The fixation was done immediately, but I admit Mr. Coats's imputation about imperfect fixation; I think formalin does not fix the retina properly; also, the section from the case of amaurotic family idiocy was cut in paraffin. The cerebral degeneration specimen was cut in celloidin, and was fixed in Zenker's fluid. The fixation in that case seemed to be fairly good, and I think the histological appearances can be fairly well relied upon, except, perhaps, in regard to the slight œdema in the internuclear layers. The ganglion cells showed degenerative changes just as in the sections of amaurotic family idiocy, and the rest of the retina showed more advanced degenerative changes probably secondary to the ganglion cell changes. As regards the relationship between the two diseases, I think they are closely allied. The pathological changes in the brain and in the retinal ganglion cells are identical. The difference in the ophthalmoscopic changes in the macular region are, in my opinion, insufficient to separate the two diseases from a pathological standpoint.

MR. TREACHER COLLINS: I think a better title for Mr. Coats's paper would have been "The Causes of Opacity of the Retina," because it deals with a

¹ Leber admits the case of Rubert, and believes, therefore, that the occurrence of œdema is proved (Graefe-Saemisch "Handbuch," 1914, part II, vii, chap. x, p. 226). The fixative, however, was Kaiserling's fluid.

much larger subject than the cause of the ophthalmoscopic appearances in amaurotic family idiocy. We meet with opacity of the retina in several different affections: in amaurotic family idiocy, in embolism of the central artery, in what is called "retinitis," in some cases of detached retina, in the cases of concussion injury of the eye, when we find what is called commotio retinae. Opacity of the retina must be either due to changes in the elements of the retina itself or to some exudation into the retina. In amaurotic family idiocy the debatable point is as to whether the opacity is due to effusion into the retina or to change in its ganglion cells; and the author of this paper has demonstrated that it is due to the latter. In the cases of sudden obstruction to the arterial blood supply to the retina, most text-books say the opacity is due to oedema. But Hancock pointed out a few years ago how very improbable it was that obstruction of an artery would produce oedema, and how the changes in the retina corresponded with the distribution of its ganglion cells. Two years ago, in removing a tumour of the orbit from behind the eye, I had to sever the optic nerve and structures round it. We looked into the eye with the ophthalmoscope for some days afterwards, and a white appearance of the retina, like that in embolism of the central artery, appeared. Then an ulcer of the cornea formed, and at the end of eleven days, at the patient's request, I removed the eye. It was examined pathologically, and an account of the findings was given by Dr. Komoto at the last meeting of the Ophthalmological Society. In that eye the pathological changes in the retina were very instructive. There was no sign of oedema, but there was marked coagulation necrosis, most intense at the posterior pole, and diminishing in intensity anteriorly. In the anterior parts of the fundus a red reflex was seen with the ophthalmoscope, the white opacity being round the maculo-papular area—that is, where the coagulation necrosis was most intense. The degenerative changes gradually became less intense as one passed forwards, the ganglion cells evidently being the first structure to disintegrate. We get opacity of the retina in "retinitis," which is due to effusion into the retina. There is plenty of pathological evidence that in albuminuric retinitis it is due to an effusion into the retina. In detached retina it is usually only in the late stages that the membrane becomes opaque, and in these late stages one finds evidence of oedema. In detached retina the opacity comes on late and remains permanent. In embolism of the central artery it appears early, and is only temporary. In the cases of commotio retinae the cause of the opacity, I think, is doubtful. I believe it to be very similar to that met with in embolism of the central artery, and that it is a change in the ganglion cells, which passes away quickly.

Mr. J. HERBERT PARSONS: I have no desire to go over the whole subject with which this paper deals to-night, but will confine myself to a few remarks on amaurotic family idiocy. I agree with most of what Mr. Treacher Collins has said. I do not agree altogether with Mr. Coats, because I do not think

that œdema and the other pathological changes under consideration are so incompatible as they seem to some persons. I cut some sections from a case of amaurotic family idiocy. There were large œdematous spaces, similar to those in Mr. Mayou's specimen, and these, I do think, cannot be explained as merely the result of bad fixation. If that were so, one would expect to find such conditions frequently in the neighbourhood of the macula, which is not the case. If a good fixative like Zenker's fluid is used, possibly there may be an actual diminution in the fluid, thus leading to a wrong idea as to the condition of the retina before fixation. In all probability, both conditions occur. I think "coagulation necrosis" is not at all a good name; in fact, the nomenclature of the whole subject needs elaboration. It is better to say that the opacity is produced as a result of disintegration of tissues, owing to the cutting off of the blood supply or some other cause. But in the vast majority of cases where this disintegrating process goes on, the mere fact that such disintegrating tissues are present, and have to be absorbed, practically always results in an increase of the lymphatic fluid in the neighbourhood, which is one form of œdema as one uses that term. The other point to which I would draw attention—though it has been somewhat dragged into this discussion—concerns the paper by Dr. Batten and Mr. Mayou. I think that was a most admirable paper, and I listened to it with the greatest interest; but in some respects it was a retrograde step. In my opinion amaurotic family idiocy is one of the most sharply defined clinical entities which we can encounter; and this appears to hold true in the light of pathological and neurological investigation, so far as that has been carried. In the literature it has been customary to mix up cases of amaurotic family idiocy with other forms of familial degeneration, especially those associated with macular changes. In most of those cases the macular changes have been quite different; there have been pigmentation and other changes which do not occur in amaurotic family idiocy. In the paper to which I refer, an attempt was made to establish amaurotic family idiocy as belonging to that group of cases in which children go on living to 15 or 16 years of age and then develop a clinically different condition. Though the cases may be explained on the lines which Dr. Batten intimated, I think that in the present state of our knowledge it is regrettable to merge them with amaurotic family idiocy in the way he urges should be done.

Mr. R. AFFLECK GREEVES (in reply): All I have to say is that I think bad fixation, and more especially formalin fixation, will cause appearances in normal retinæ which simulate those seen in œdematous retinæ fixed by more satisfactory methods. I have had a good deal of experience with Zenker-fixed retinæ, and I believe that one can detect even a slight degree of true œdema after fixation by this method.

(February 2, 1916.)

**On Superficial Linear Keratitis, together with an Account of
the Pathological Examination of Two Affected Eyes.**

By W. T. HOLMES SPICER, F.R.C.S., and R. AFFLECK GREEVES,
F.R.C.S.

(I) CLINICAL INTRODUCTION BY MR. W. T. HOLMES SPICER.

THIS is, so far as my experience goes, a rare condition; but inasmuch as it presents appearances which have been remarkably uniform in all the examples which have come under my personal observation, it must be regarded as a clinical entity. I have had the opportunity of getting two specimens of whole eyes for microscopic examination, and I thought it would interest the Section to hear the clinical report and pathological examination together. The pathological examination has been made for me by Mr. R. A. Greeves.

It is possible to divide the cases at once into two groups: in the first group are the simple cases, which get well speedily and do not recur; in the second group the clinical features are identical with those of the first group, and to a great extent they make the same recovery, but are followed by a series of relapses, resembling those of relapsing bullous keratitis, going on for months and years (in one of my cases for sixteen years), and lead to great damage to the eye, even to loss of sight.

CLINICAL CHARACTERS.

The clinical features are sudden onset with pain and congestion in an eye, the appearance of a number of superficial ridges of epithelium in the cornea raised from the general level of the corneal surface, for the most part vertical in arrangement, grey in colour with tapering ends, not reaching to the limbus. Under magnification, the ridges are double contoured with a comparatively clear centre, they are denser and thicker in places, giving an appearance of nodes at more or less regular intervals. As a rule, the ridge does not as a whole stain with fluorescein, but punctate staining along its course is common, especially so over the nodes.

The affected eye is, as a rule, very slightly or moderately congested. No iritis and no corneal precipitates are present.

One remarkable feature, which is almost always found during at least some period of the attack, is a softening of the eye, so that tension falls to -1 and often to -2 ; during this time of diminished tension the vision is greatly impaired, and in one case it dropped to $\frac{2}{60}$. The attack itself lasts from a few days to a week or longer, and as the ridge disappears the vision recovers and the tension becomes normal again; sometimes the diminution in tension lasts only a few hours. The only condition left behind after each attack is a slight greyness or visibility of the corneal epithelium along the track; if the attack does not recur this greyness passes, but where a succession of attacks goes on for months a permanent greyness is established, which in the worst cases makes the corneal surface opaque.

Pain varies, but in the worst cases it is considerable, especially with the diminished tension, and the patient becomes much worn and exhausted with the pain and worry due to the eye. There is no anaesthesia of the cornea.

It is mostly young adults who are affected. It cannot be said that they are remarkable in any way, but the worst cases impress one as being poorly nourished and thin, and as their general nutrition improves, the state of the eye also ameliorates. On the other hand, part of their general poorness of condition is due to the wearing nature of the eye trouble.

TREATMENT.

To this end nothing has been found to be of real or permanent value. The immediate attacks yield to ordinary treatment on general lines, but the recurrences are not prevented by anything I or others have used.

Among the measures that have been employed are heat in various forms, antiseptic lotions, aristol, pyridine, atropine, eserine, dionine, and massage with mercurial ointment, absolute phenol and absolute alcohol applied to the cornea. Also peritomy, iridectomy, radium emanations from Bath water, radium ionizations, and change of air, including residence in Switzerland.

NATURE AND AFFINITIES.

The disease is clearly epithelial, with an underlying neuropathic element, as evidenced by the diminished tension.

In anatomical situation it has something in common with superficial punctate keratitis, in which the points are analogous to the nodes in the cases under consideration, but the resemblance goes no further. Its appearance is somewhat like that of the dendritic ulcer, but the lines are multiple, are much narrower and straighter in their course, and do not possess the bud-like outgrowths from the central stem. It also differs in its greater resistance to treatment and in its persistent recurrences; moreover, the dendritic ulcer is not as a rule accompanied by great lowering of tension.

In many respects it is like the recurrent bullous keratitis or relapsing abrasion; but it is not always the result of an injury, and its linear form as well as its peculiarity in regard to ocular tension clearly separate it from this disease.

It is most probable that diminished tension is the essential feature, and that the lines are produced by a kind of crumpling or folding of the relaxed Bowman's membrane. This view of it was first suggested by the behaviour of one of the earlier cases that I saw, in which an injury to the cornea treated by a compress was followed by the appearance of lines. These seemed to be the result of pressure, as they disappeared soon after the use of the compress was discontinued. But the lines may appear without any pressure being applied.

It is in favour of the neuropathic element that one at least of my patients complains of the eye feeling numb, as if it were asleep, when the attacks are coming on, although there is no loss of sensation. The diminished tension points either to a toxic action on the secreting epithelium of the ciliary body or to a reduction of secreting activity from altered innervation to the internal secreting areas of the eye.

The evidence of toxic absorption is not present, as neither iritis nor cyclitis—that is, corneal precipitates—are ever seen during the course of the disease.

NOMENCLATURE.

I have preferred to make the name as far as possible descriptive. The affection is inflammatory, it is linear, and it is superficial. The terms "reticular," or "lattice," or "trellis keratitis" have been applied to other affections of the cornea, clearly not of an inflammatory or acute nature.

I have looked through the references to similar affections, and the only case I have come across corresponding at all with my own is recorded by L. Caspar in the *Klinische Monatsblätter für Augenheilkunde*

for 1903 (p. 289), in which a week after an injury a number of superficial lines appeared in the centre of the cornea not connected with the injured spot, with knob-like thickenings which stained; the lines disappeared and left no mark. Caspar considered the cause to lie in a flattening and distortion of the cornea, giving rise to the formation of longitudinal grooves.

CASES.

Case I: Superficial Linear Keratitis proceeding to Loss of One Eye and subsequent Involvement of the other.—C. H., aged 33, came under the care of Mr. Gunn, at Moorfields, on December 28, 1901: The left eye began to be troublesome at the age of 18; she had attacks at intervals of three or four months at that time, but they became less frequent after. The last attack before the present one was four years ago. She was then treated at the Oxford Eye Hospital as a case of dendritic ulcer. Her general health was fairly good. Urine, 1024, acid, no albumin, no sugar. She was rather thin, but took her food well, and had no indigestion. Vision: R. $\frac{6}{12}$, + 2 = $\frac{6}{8}$; L. $\frac{6}{60}$, not improved. Visual fields full. Tension normal. There is moderate ciliary congestion; a general superficial haziness of the cornea with blurring of details of deeper parts. The pupil dilated fully with atropine. Details of left fundus not well seen, but nothing abnormal remarked. The lenses are clear. There is no staining with fluorescein. On examining the cornea fully, a number of grey lines can be seen running, generally in a vertical direction; the lines are raised above the surface into a very distinct ridge, so that in some places the impression given is that of an elongated vesicle or of a chain of vesicles. The whole appearance suggests a surface affection, and the lines are apparently made up of epithelium only; where the vesicular appearance is not marked, the lines appear double-contoured, that is, of tubular structure. Between these clearer parts are certain nodules denser than the rest of the lines. Besides these broad superficial lines with raised surface, the rest of the cornea is covered with very delicate grey lines running in the same direction as the broad ones, that is, vertically; in places, these merge into the broad lines or disappear behind them; all the lines taper off to nothing at the upper and lower periphery. The faint lines suggest that they are old tracks which have healed. Patient discharged on February 4, 1902. October 27, 1902: Was re-admitted with the eye in the same condition. She was treated locally by hot applications and by atropine; later, by eserine, without effecting any marked improvement. December 24: Iridectomy upwards was done; no benefit resulted. Tension was normal. January 17, 1903: The sclerotic was cauterized all round just outside the limbus. This resulted in an improvement of the symptoms, the irritation, photophobia, and weeping being a good deal lessened; but there was not much effect on the corneal condition at once, although the vision improved to $\frac{6}{18}$. April 27: A relapse. No staining of

cornea, but it was painted with phenol. February 10, 1904: Recurrence three weeks ago; there has been very great pain. The congestion is very great, the cornea rough and covered with vertical lines (fig. 1). Anterior chamber shallow. Iridectomy coloboma upward; pillars of iris free. February 18: It was decided to remove the eye, since there was no prospect of cure, and the



FIG. 1.

C. H., aged 33. Drawing made February 10, 1904. Iridectomy coloboma upward. Raised, vertically running lines; in places double-contoured, with thicker parts at intervals. Also fainter lines, vertical in direction, all over the cornea.

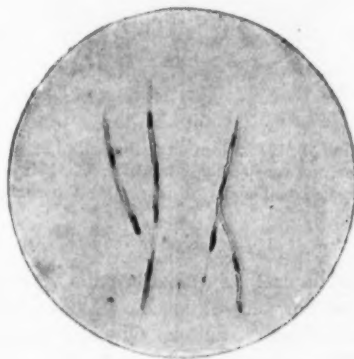


FIG. 2.

C. H. Second eye. Raised lines grey, double-contoured, with nodules in places of greater density but not of greater thickness.

patient's life was being made useless and irksome. October 25, 1904: The patient was re-admitted, as the vision in the right eye had been getting dim for a month, and she had been having much headache in the frontal region.

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Right eye: Lid swollen; general haze of cornea with dense striated opacities and nodes; the lines suggest double contours, and the nodes are whiter and broader than the rest of the lines (fig. 2). Sensation rather dull; anterior chamber normal. Pupil $3/4$ dilated. Iris bright. Vision: Sph. + 2, cyl. + 1.5, $1\frac{6}{12}$. November 22, 1904: Discharged. No further account of the patient.

Case II: Superficial Linear Keratitis with Loss of one Eye and subsequent Involvement of the Second Eye.—Miss D. T. S. was sent to me by Dr. J. Burdon-Cooper on May 13, 1914. She was under the care of Dr. Wilson Smith. The left eye had begun to trouble her in October, 1913. It began as faint grey lines, having a generally vertical direction, at first without any other ocular disturbance and with little congestion. The tension was much reduced. The treatment she had received at various times consisted of antiseptic lotions, drops, and ointments; atropine, aristol, pyridine, applications of absolute alcohol,



FIG. 3.

Miss D. T. S. May 13, 1914: Six vertically running white lines near the surface of the cornea, not very straight, tapering at their ends, with a double contour and with dense broad nodes at intervals; the surface is raised into a ridge over the lines.

massage. Nothing was of use. The eye became very painful, lost all useful sight, and was affecting her general health. It was excised in April, 1914. The other eye had already begun to suffer. One character common to both eyes was the marked reduction in tension. May 13, 1914: When I saw her there was moderate ciliary congestion, a painful, somewhat tender eye, with a very depressed state of mind and health, as she was apprehensive of losing her remaining eye. There were five or six vertically running sinuous grey lines near the surface of the cornea, with tapering ends, and accentuated edges almost forming double contours. At intervals along the lines there were nodes broader and whiter than the lines generally; the surface epithelium was distinctly raised into a ridge over the lines (fig. 3); no precipitates; no other corneal changes;

no iritis. Tension -1. About a week after the patient left she was given radium emanations from Bath water, the gas being passed over the eye through an eye-cup with an entrance and exit. The gas came from a cylinder filled with the emanations taken from the surface of the water. From this time there was rapid recovery, but she thinks the eye had begun to improve before she had the gas, as she had been able to see a gas globe against a white curtain, and she had not been able to see any forms before. She was also fed up as much as possible, and among other things she took half a pint of cream daily. July 3, 1914: At this date there was no congestion, the corneal surface was quite smooth, there was no opacity, and the lines had gone; there were no keratitic deposits and no iritis. The tension was normal.

$$\text{Right vision } \begin{array}{r} +0.75 \\ +0.5 \end{array} = \frac{6}{6}.$$

November 12, 1915: In reply to my inquiries, I heard from Dr. Wilson Smith, of Bath, who reported: "She continued to make progress to complete cure and remained well for some months. She got caught in Switzerland at the beginning of the War and had some difficulty in returning, and shortly afterwards her eye relapsed. I again tried radium emanations with slight improvement, but without a cure. I substituted radium ionization for the last several months in the summer, I think with some slight benefit. February 2, 1916: On examination to-day I find the vision $\frac{6}{36}$. Retinoscopy—

$$\begin{array}{c} +2 \\ + \\ +4, \end{array}$$

but no improvement in vision with any glass. There are now several superficial grey lines in the cornea with pronounced nodules all along them, and with a few punctate elevations of epithelium, but with no general raising of the surface. Tension -1. Pupil dilates well. Fundus normal. She has no pain, but the eye feels quite numb when an attack is coming on, like a limb that has gone to sleep."

Case III: Recurrent Attacks of Superficial Linear Keratitis with Recovery between the Attacks.—W. C., aged 17. Engaged in weighing cartridges at Woolwich. January 28, 1916: This case came under Mr. J. H. Fisher's care at Moorfields Hospital; I have to thank him for letting me use it. Three months ago the patient felt a pricking in the left eye. At the Arsenal surgery he was told there was a piece of brass in the eye. It was removed, and the eye seemed all right afterwards and he returned to work; two weeks later the eye started pricking again, and he was given drops and returned to work. He has now had about four attacks, and the last one is the worst he has experienced. There is little or no fear of light, and but slight injection of the left eye. There is a faint grey, trident-shaped opacity of the surface of the cornea, more dense at the lower part and showing a slight disturbance at the surface; at the lowest part, the base of the trident, the greyness extends over a considerable

area. In the centre of each of the grey lines there is a small raised ridge of epithelium, not perfectly continuous, and not staining continuously with fluorescein. The whole appearance is as if there were a delicate mycelium growing or lying on the surface of the cornea; the lines, however, cannot be moved, and, by careful examination, can be seen to be a breach in the epithelium. There is no appearance of nodules; but the breaks in the staining line show that certain parts of the lines are denser than others (fig. 4). The tension of the eye is normal; there is no anaesthesia. The pupil dilates well.

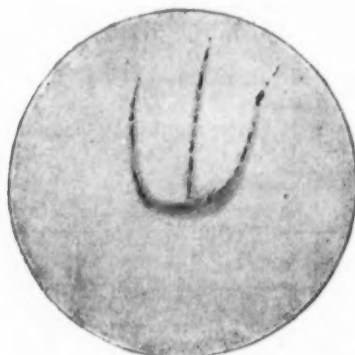


FIG. 4.

Case IV: Superficial Linear Keratitis after Removal of a Foreign Body.—June 23, 1905: A. T., aged 16, came to St. Bartholomew's Hospital with a piece of steel embedded in the left cornea. This was removed by Mr. R. Foster Moore, who was then house surgeon. Some atropine was used, and the eye was bandaged. Two days later, when I saw him, vision was reduced to $\frac{2}{60}$, and the tension of the eye was -1. There were traces of the abrasion at about 7 o'clock, midway between centre and periphery, stained brown with rust, and not at all deep in the cornea. From this spot there were about four lines extending mainly up and in, across the cornea; they were epithelial ridges, and when the light caught them obliquely, they could be seen to be definitely raised above the general level of the cornea; looked at directly, they were grey in colour and opaque. The corneal epithelium generally was slightly oedematous. June 29: Four days later the lines had disappeared.

Case V: Superficial Linear Keratitis; Recovery.—W. A., aged 16, lift-boy. Seen at St. Bartholomew's Hospital. January 30, 1913: The right eye had been watering and "gummy" for two weeks; it had been red and painful; he had had no injury; was in good health; never had bad eyes before. Vision: Right $\frac{6}{60}$, J.20, not improved; left $\frac{6}{60}$, J.1; tension normal. In the right eye are many vertical grey striæ, quite superficial, forming prominent ridges on the

surface of the cornea reaching to the limbus above, and below ending in a crescent-shaped opacity staining with fluorescein. Otherwise the cornea and iris are normal; tension -2. He was taken into the hospital, kept in bed, the eye treated with fomentations and atropine. In two days he was much better. A week later, on February 8, 1913, the cornea had cleared, there was slight ciliary congestion, and a little roughness of the cornea below. His vision was $\frac{6}{9}$.

Case VI.—Another case in a man was shown me by Mr. P. Flemming, under his care at Moorfields Hospital. No injury. There were six vertically running ridges in the cornea with broken, swollen surface, looking as if the eye had been scratched by a brush. At intervals there were nodes of greater density. His eye had been bad two weeks. With ordinary treatment he made a good recovery.

Case VII.—Mr. J. Cole Marshall showed me a case of a boy under his care who had had a blow on the eye without a wound three weeks before. There was moderate congestion, and four superficial grey lines raised above the surface of the cornea, with characteristic double contours and nodes. The tension was -1. He did not respond rapidly to treatment, and discontinued his attendance.

(II) PATHOLOGICAL EXAMINATION BY R. AFFLECK GREEVES, F.R.C.S.

The following appearances were shown by microscopical sections of the eye in Case I. The sections, which were taken through the cornea at right angles to the direction of the linear opacities, show that the raised lines on the corneal surface are due to ridges in Bowman's membrane. These ridges vary in appearance in different places, but they are always characterized by the presence of new fibrous tissue in the immediately underlying substantia propria.

Some of the ridges are narrow and sharp, Bowman's membrane being raised up into a single well-marked fold (fig. 5), while the majority are broader and show a double elevation (fig. 6). In some parts they broaden out into a series of ridges, with a strongly marked layer of underlying fibrous tissue (fig. 7). In some of the latter, Bowman's membrane shows numerous bends and convolutions, and its continuity is sometimes broken (fig. 8).

Over most of the more raised elevations the corneal epithelium is absent in the sections, but this I believe to be an artefact, the epithelium having been rubbed off in the course of preparation, for in some sections a stripped-off thin strand of epithelium can be seen still hanging on to the more normal epithelium at one side or the other of the prominence.

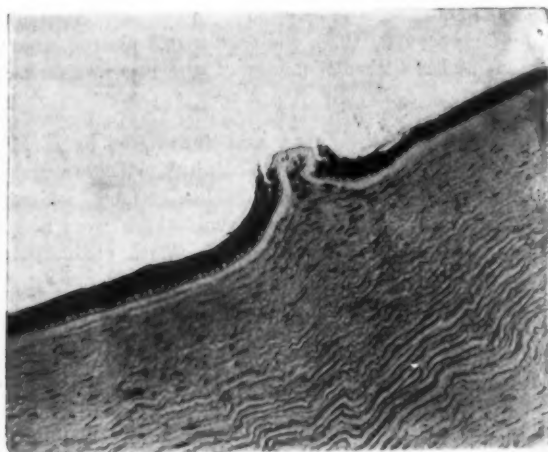


FIG. 5.

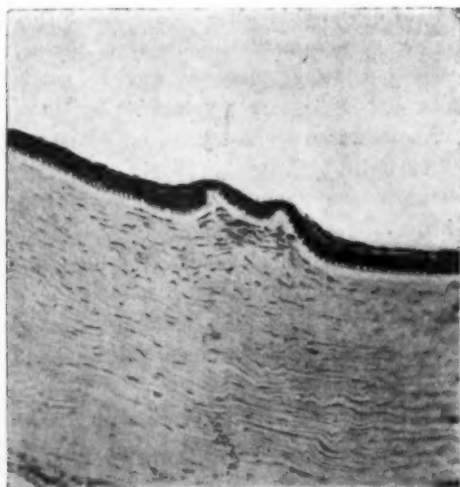


FIG. 6.

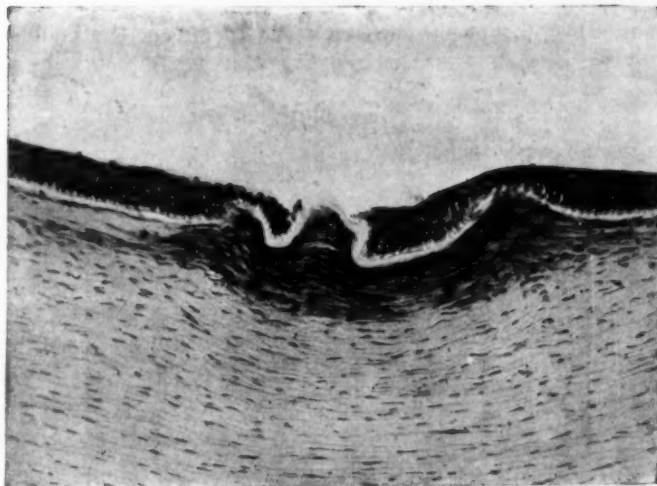


FIG. 7.

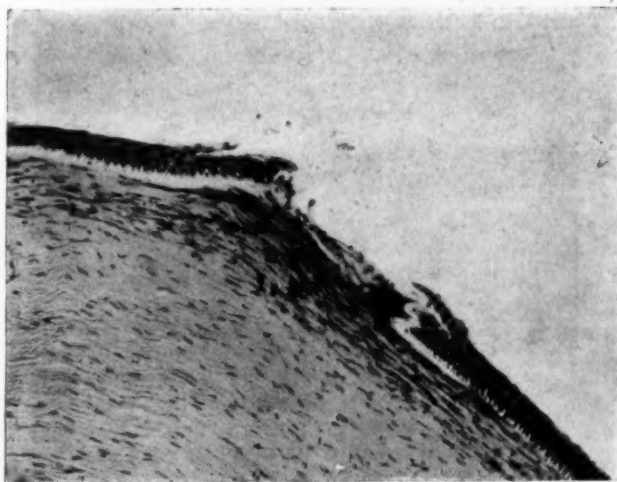


FIG. 8.

50 Spicer and Greeves: *On Superficial Linear Keratitis*

These strands consist of at most one or two layers of very much flattened epithelial cells.

Some underlying fibrous tissue is always present, although it varies in amount. Fig. 9 shows the extreme end of one of the double ridge

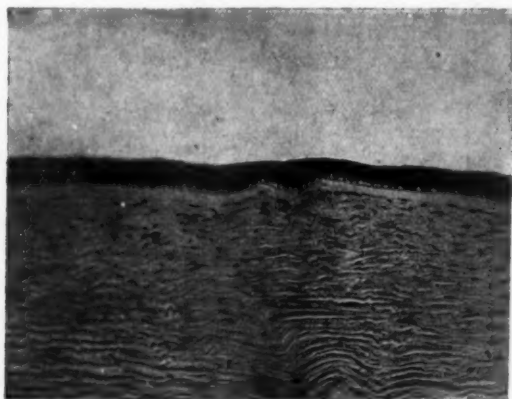


FIG. 9.

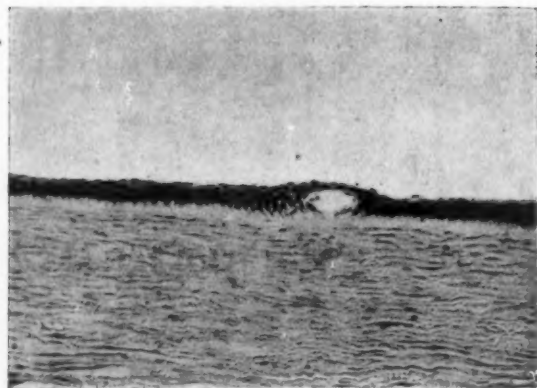


FIG. 10.

lines—even here a small amount of fibrous tissue can be made out. This fibrous tissue can only have been produced by the activity of the corneal corpuscles in the substantia propria, since in no case has vascularization of the tissue been found.

A possible suggestion is that the formation of fibrous tissue under Bowman's membrane precedes the wrinkling of the membrane, and that the latter is caused by the contraction and increase in quantity of the fibrous tissue. The epithelium over the rest of the cornea shows varying degrees of œdema. In some places bullæ are present, under

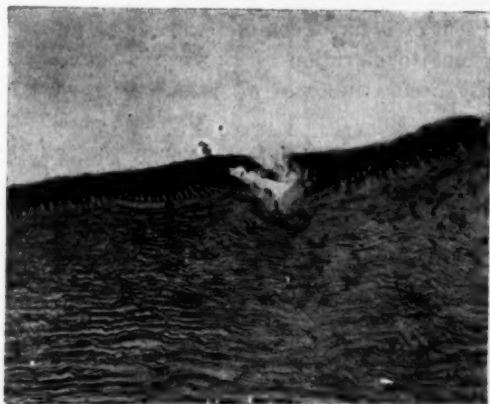


FIG. 11.

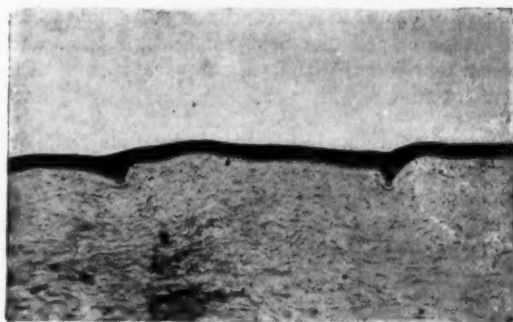


FIG. 12.

some of which Bowman's membrane is unchanged (fig. 10), while underneath others there are definite depressions in the membrane (fig. 11). There is no evidence that the bullæ are precursors of the ridges, although this is, of course, a possibility.

The eye in Case II (Miss D. T. S.) had undergone a good deal of drying before it was fixed, with the result that the staining of the cornea was unsatisfactory and that consequently good photographs could not be obtained. Ridges in Bowman's membrane with underlying fibrous tissue could, however, be made out in the sections. No vascularization was present.

For the sake of comparison, I will show a section through a cornea which was the seat of a fairly common variety of linear opacity, due to wrinkling, but not to elevation of Bowman's membrane. This form of linear opacity occurs in cases of extensive scarring of the cornea, the clear residue of the cornea being the seat of the linear opacities. The section (fig. 12) is taken from a case of that kind. The eye was beginning to shrink and had a definitely lowered tension. It will be seen that the epithelium fills up the hollows caused by the wrinkling of Bowman's membrane, and that the corneal surface is almost level.

DISCUSSION.

MR. JAMES COLE MARSHALL: I have the notes of the case to which Mr. Holmes Spicer referred. The patient was a man who had his eye knocked by a cow's horn, and he presented all the features which Mr. Spicer pointed out; but in addition to the superficial lines there was also much œdema, and a good deal of spotted superficial keratitis. There was generally very low tension in the eyeball, usually -1, but nothing else was specially noteworthy about the eye. With regard to treatment, the only thing which did good was atropine at the beginning and dionine, 5 per cent., instilled once daily. After attending three or four months the patient gave up, and I could not find out any more about him.

MR. J. B. LAWFORD: I should like to ask Mr. Spicer one or two questions. In all his sketches I think the striæ appear to be more or less central. Did they usually begin centrally or peripherally? In one case under my observation the stalks seemed to come from near the lower border of the cornea and to spread upwards. Another point is as to sex-incidence. The only case I have seen was in a female, and two or three of Mr. Spicer's cases were females. Can he tell us the sex proportion?

MR. D. L. DAVIES: I feel some hesitation in suggesting a line of treatment for these patients. I wonder whether in these cases of neuropathic origin the exhibition of thyroid might do good. Last summer I had a case of dendritic keratitis in a trooper, aged 20, who had had many attacks before. I knew the difficulty of treating these cases and I decided to try thyroid, and he certainly improved very rapidly. I gave him in addition the usual local treatment—atropine and fomentations—so long as the pain persisted, and in

three weeks he was cured, although, as he told me, all his previous attacks had lasted two months.

Mr. HOLMES SPICER (in reply): Mr. Lawford has asked me about the point of origin of the appearances. So far as I have seen they always appeared in the centre, and then have extended across towards the periphery. In Mr. Fisher's recent case it appeared in the upper part of the cornea, but extended to the centre and perhaps below it. With regard to the sexes, the two worst cases I have seen have been in women; the transitory conditions have been, in each case, in males. I thank Mr. Davies for his suggestion about thyroid treatment: I will bear it in mind.

(February 2, 1916.)

Double Cataract following Experimental Total Thyroidectomy in a Dog.

By WALTER EDMUNDS, F.R.C.S.

THE dog, having been fed for several days on milk only, was subjected to total excision of the thyroid gland and the parathyroids. He was kept on a diet of milk only, to which 30 gr. of lactate of calcium were added daily. He remained well till the nineteenth day, when he appeared dull, did not stand up, and had tremors; an intravenous injection of 8 gr. of lactate of calcium was given. The next day he was better and had no tremors. The day after that he was well, but four days later the symptoms returned; another injection of lactate of calcium was given, he was immediately better and remained so the next day, but the day after that he had a return of symptoms, when a third injection was given; the next day he was well, and remained so for nine months, when he had a fit, but he had only one fit, and remained well for one year and five months after the operation, then it was noticed that he was somewhat thin, and also bald on the inside of his thighs and to some extent on his head; he was ordered cream in addition to that contained in the milk, which was his sole diet; he regained his condition, so that after a time the cream was stopped, and he remained well for another six months, except that he was noticed to be losing his sight from double cataract.

The late Mr. George Coats kindly saw the dog, and reported as

follows: "The lens opacity does not seem to be due to any disease of the eye itself; the pupil is active and the dog seems to have perception of light, although it cannot avoid obstacles. I should think the cataract is undoubtedly due to general nutritional changes connected with the removal of the thyroid and with the fits which I believe followed it. This association of cataract with extirpation of the thyroid and of 'tetany' is known in the case of man."

A few days later the dog was weak, dull, and had tremors; an intravenous injection of calcium lactate was given, and the next day the dog was much better, but in a few days he died, no doubt as a late result of the operation performed just two years previously. A post-mortem examination was made: the pituitary was large, in all probability enlarged; it was very vascular, the secreting cells were very large, and some contained two nuclei.

The eyes were handed to Mr. Coats, who reported: "I do not think any change can be detected in the lens epithelium either in horizontal or coronal sections; compared with the normal dog's eye, I should say there was a certain amount of infiltration of the ciliary processes, with loosening and tightening of the pigmentation of the outer layer of the epithelium; the inner layers of the stroma show a little of the same thing. The aqueous contains a good deal of fibrinous coagulum, perhaps indicating an alteration in composition. These changes may have to do with the cataract, or may possibly be due to slight inflammation prior to the animal's death. Before coming to a definite conclusion it would be necessary to know whether they are constantly present in cases of thyroidectomy, and as regards the pigmentation it would be necessary to make a comparison with the condition in animals of the same breed, and, if possible, of approximately the same age. The cataractous changes themselves do not belong to any very special type. They are most developed in the deeper layers of the cortex, where senile changes most often begin in the human eye. They are not of distinctly lamellar type. In one eye there has been a rupture of lens capsule on both sides just behind the equator (actual rupture is not shown in this section), with escape of some lens substance and formation of capsular cataract on the outer aspect of the capsule; but I think this must be secondary, and caused most probably by swelling of the lens as it became cataractous. It is in the region of the insertion of the posterior fibres of the zonula where probably there would be some strain, while the capsule is thinner there than at the insertion of the anterior fibres."

REMARKS.

Although in this dog both the thyroid and the parathyroids were removed, its symptoms and death were probably due mainly to the removal of the parathyroids, as tremors, which occur after this operation, were present shortly before death. It would seem that the cataracts should be attributed to the same cause.

There are on record at least two cases of goitre in the human subject in which an operation on the thyroid had been carried out, and in whom cataract followed. In both cases there was tetany.

Case I.—Recorded by A. Westphal. Patient was a woman who, since 1881, had suffered from an enlargement of the thyroid gland. In May, 1895, the gland was completely removed by operation. The second day after the operation there were symptoms of tetany. In January, 1900, she was admitted to hospital. She had then an apathetic expression; she suffered from tetany and epileptic fits, and also had double cataract. The symptoms point to the removal of the parathyroids as their main cause.

Case II.—Recorded by A. Schiller. A woman, aged 36, from the age of 22 had an enlargement of all the lobes of the thyroid; they now caused difficulty of respiration and aphonia. An operation was performed by Czerny in June, 1890. The goitre was to a great extent removed, but part of one lobe was left; the trachea was found compressed. Two days after operation tetanic spasms occurred, especially in the lower half of the body; a few days later there appeared albuminuria without casts, œdema of the hands and mental dullness. The following winter the tetany continued, and cataract appeared in both eyes; the right eye was operated on. The next summer there was cachexia, the skin was dry, and the hair and nails fell out. In the autumn the nephritis became acute, and the patient died.

The tetany here spoken of is probably distinct from the carpo-pedal spasm of children, sometimes called infantile tetany; but the administration of parathyroid gland is said by Bliss to have been found of benefit in some cases of this disease.

With respect to treatment in dogs which have been operated on, the best treatment seems to be to give calcium by feeding them wholly on milk (which contains much calcium), or, if the symptoms are severe, by the intravenous injection of the lactate of calcium; this with me has proved better than the administration of parathyroid gland.

In tetany, following a thyroid operation in a woman, Halsted treated the symptoms (which he attributed to removal of parathyroid) with the parathyroid from the ox, and with much benefit: "the effect of the parathyroid exhibition was almost instantaneous and most marvellous."

Hertz also has recorded the case of a patient who, a few months after an operation on the thyroid for goitre, became restless, nervous, sleepless, tremulous, and thin. Treatment with parathyroid was successful: "The remarkable effect of treatment with dried parathyroid gland after everything else had failed makes it very probable that the group of symptoms from which the patient suffered was the result of parathyroid insufficiency."

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DISCUSSION.

Mr. J. HERBERT PARSONS: I should like to ask whether Mr. Edmunds has seen interstitial keratitis in these thyroidectomized animals. In 1891 I experimented on a series of animals for physiological purposes when I was investigating nitrogenous metabolism after thyroidectomy. We did not know much about the parathyroids in those days, but after total thyroidectomy the dogs promptly died of tetany. In some, only part of the thyroid was excised, and the animals could be kept alive for a considerable time. One of these developed typical interstitial keratitis, a condition which is not very uncommon in dogs, especially in those highly bred. I do not know whether the occurrence was a mere coincidence, or whether Mr. Edmunds, who has had more experience in thyroidectomy in animals than anybody else, has also noticed it.

Mr. J. HERBERT FISHER: I should like to ask Mr. Edmunds a question with regard to myxœdema, which disease, I presume, is due to atrophy of the thyroid gland; but I am ignorant as to the condition of the parathyroids in myxœdema. It must be generally known whether myxœdema is often accompanied by cataract or not. Personally, I can only recall one case of cataract in a myxœdematous woman, and in that case I operated, with a very favourable result; there were no bad symptoms, either immediate or subsequent. I am not sure whether the parathyroids are active or inactive in myxœdema.

Mr. WALTER EDMUNDS (in reply): I remember seeing ulceration of the cornea in some of these dogs I experimented on, but I do not know its exact nature or cause; I do not think it was interstitial keratitis. In myxœdema the parathyroids are said to be normal; myxœdema is attributed solely to thyroid insufficiency. With regard to cataract occurring in myxœdema, I know of one such case, but it does not follow that the cataract was due to the myxœdema.

Section of Ophthalmology.

President—Mr. PRIESTLEY SMITH, F.R.C.S.

(March 22, 1916.)

Disturbances of Vision from Cerebral Lesions, with Special Reference to the Cortical Representation of the Macula.

By W. T. LISTER,¹ F.R.C.S., Colonel A.M.S., C.M.G., and
GORDON HOLMES, M.D., Lieutenant-Colonel R.A.M.C.

IN our work in the base hospitals in France during the past eighteen months, we have observed a very large number of cases in which vision was affected by the lesions of various portions of the optical system, but time and opportunity have not permitted a careful and complete examination of all. Consequently, in many cases of considerable clinical importance we possess only scanty and incomplete notes; but by selecting for thorough investigation a certain number of suitable cases we have been able to collect a group of facts that have an important bearing on the cerebral localization of vision, and more particularly on the representation of different regions of the retina in the cortex.

Owing to the conditions under which we have worked, most of our observations were necessarily made at a relatively early date after the infliction of the wound; we recognize that this fact makes possible the objection that, as our cases were mainly examined during the stage in which the effects of shock or diaschisis were still present, the visual defects we describe may have been due to functional disturbances rather than to local injuries of the corresponding cortical areas or of their centripetal fibres: but if we can show that there is a constant relation between the probable site of the injury and the form of the visual defect, it is obvious that at least certain general conclusions will be justifiable.

The fact that most of our patients were investigated in the early

¹ Consulting Ophthalmic Surgeon, British Expeditionary Force.

stages also presented certain technical difficulties; as most were confined to bed a small hand perimeter had to be employed, but in some the observations were later controlled by a McHardy perimeter. We have also employed a modification of Bjerrum's screen¹ to determine the exact relations of smaller defects. Further, since such patients tire easily and quickly, repeated examinations were often necessary. On the other hand, the fact that most of the wounds were recent has enabled us to make many interesting observations on the mode and rate of recovery of vision from different forms of injury.

Since the time of Munk, who from his experimental observations attempted to project the retina directly on to the cerebral cortex, the localization of visual function has excited much interest, but until relatively recently nothing was known of the exact limits of the visual area, or of the regional representation of the retina within it. Certain authors, especially Henschen and Dr. J. S. Bolton, have, however, discovered pathological evidence that the visual area coincides with that cortical zone of special structure which is now generally known as the area striata (fig. 1). This is the area distinguished by Gennari's line, which lies within and on both lips of the calcarine fissure, and which, even in the higher races of mankind, but to a greater extent in the lower, extends to and around the pole of the occipital lobe. There is now considerable evidence, due chiefly to the work of Henschen, that the upper half of each retina is represented in the upper parts of these areas, and the lower in the lower walls and lips of the calcarine fissures; destruction of the upper portion of the area striata on one side would consequently produce a lower quadrantic homonymous hemianopia in the opposite halves of the visual fields, and vice versa.

On the other hand, nothing is definitely known as to the correspondence between various concentric zones of the retina and the different segments of the area striata.

¹ A description of this modification may be useful, especially to those engaged in similar work. A large sheet of paper (double foolscap), covered with black cloth, is placed on a flat board and both are pinned to it. A small drawing-pin serves as a fixation point. The test object is carried on the blunt end of a steel pin, about $\frac{1}{2}$ cm. in length, which is fixed transversely across the end of a dark metal bar, so that its point projects about 1 mm. on the opposite side. The test object thus fixed on the end of the rod is moved slowly from the blind region along the screen until the patient sees it; then the carrier is simply pressed against the board so that the point of the pin penetrates the cloth and marks the paper beneath it.

A large number of observations are taken. If the distance of the point fixed from the eye is known—we generally use it at a distance of 1 m.—the record thus obtained can be transferred to an ordinary or to a special perimeter chart.

One advantage of this method is that as there are no lines or marks on the screen which can influence the patient or the observer, the records obtained by it are absolutely objective.

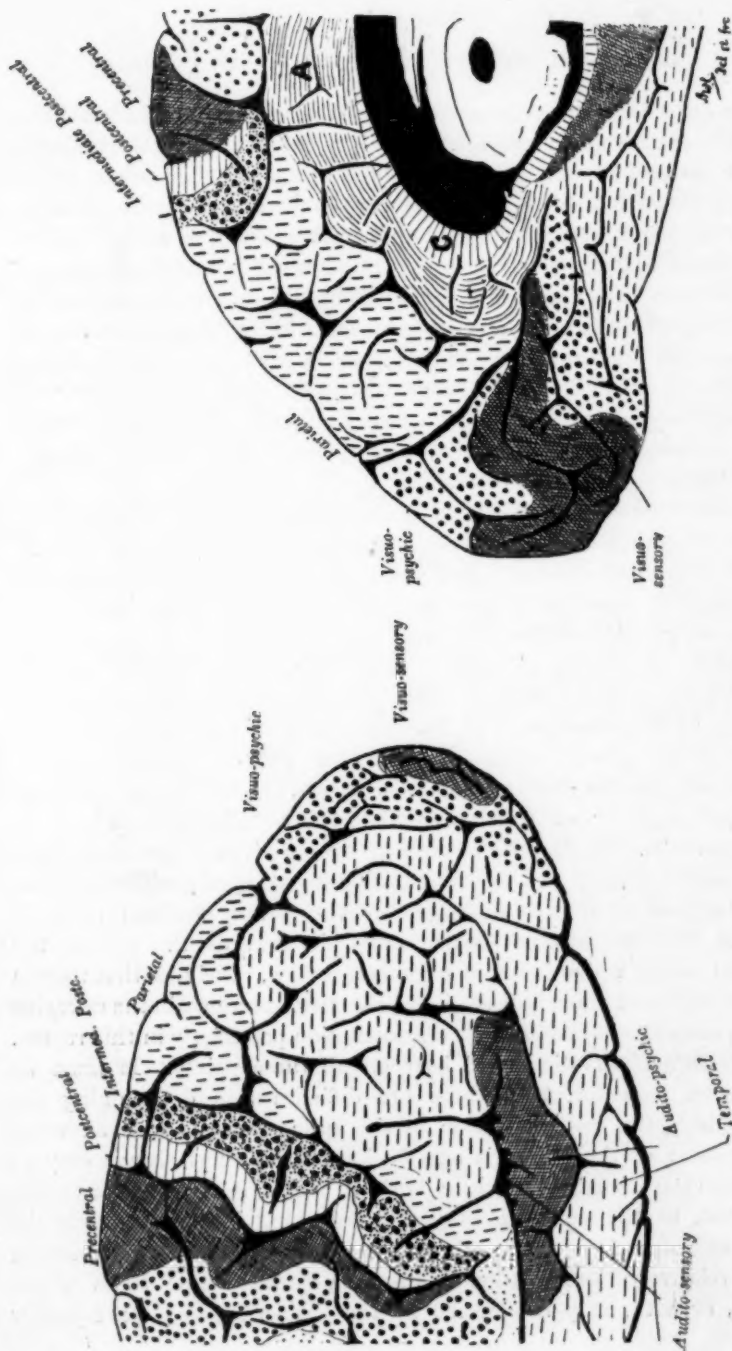


FIG. 1.

The distribution of the area striata (visuo-sensory) on the mesial and lateral surfaces of the hemisphere (Campbell).

The question of the cortical representation of the macula has for a long time excited much interest. Owing to the fact that in the majority of those cases of homonymous hemianopia due to vascular lesions which are seen in civil practice, vision is unaffected for a distance of 2° to 10° to the blind side of the fixation point, it was for a time generally assumed that the macular area is represented in both hemispheres; this hypothesis was apparently supported by the anatomical observation that at the chiasma branches of the macular fibres from each eye pass into each optic tract. But the fact that in bilateral hemianopias due to lesions of both occipital lobes central vision often persists confuted this view, and gave origin to the hypothesis that the macular centre must lie outside the area striata, or may not indeed be locally represented in the cortex (von Monakow).

Others have attempted to explain the macular escape in hemianopia by assuming that the two cortical macular centres are connected by callosal commissural fibres (Lenz, Heine), or by the hypothesis that the physiologically more highly developed macular impulses pass more easily through an incomplete block (Ronne).

Within recent years, however, many attempts have been made to determine a localized macular centre in the cortex. Henschen, for instance, at first assumed that the macular focus lies in the anterior portion of the calcarine cortex, and later that it extends along the whole floor of the calcarine fissure. Dr. J. S. Bolton, too, has recently assumed that the "calcarine cone" of the visual cortex is the anatomical basis for macular vision. On the other hand, Inouye, in his excellent monograph on the visual disturbances observed after gunshot injuries of the occipital lobes inflicted in the Russo-Japanese War, brings forward evidence showing that the macular centre must lie towards the pole of the occipital lobe; while Lenz, in a general review, concludes that though certain published cases indicate its localization anteriorly in the calcarine cortex, more are in favour of its representation posteriorly in this region.

Our own observations will bear on all these points. It must be recognized, however, that there are difficulties in determining the exact site of the essential lesion in the optic system, and even when the position of the injury is exactly known, it is not always easy to correlate the visual disturbance with it. This is largely due to the fact that, as the optic radiations, in sweeping backwards along the outer side of, and especially behind, the posterior horn of the ventricle, lie in relatively close relation to the area striata, either these or the cortex, or both, may be affected by the one lesion, and consequently

the exact cause of the visual disturbance may be uncertain. Since, however, the fibres of the radiations that reach the most posterior part of the pole are probably distributed only to the most posterior part of the calcarine cortex, visual defects associated with their injury here may bear directly on the question of cortical localization. On the other hand, an extensive lesion in the middle or anterior portion of the calcarine cortex may involve any or all of the fibres of the radiations, and the associated visual defects may give us no clue to localization.

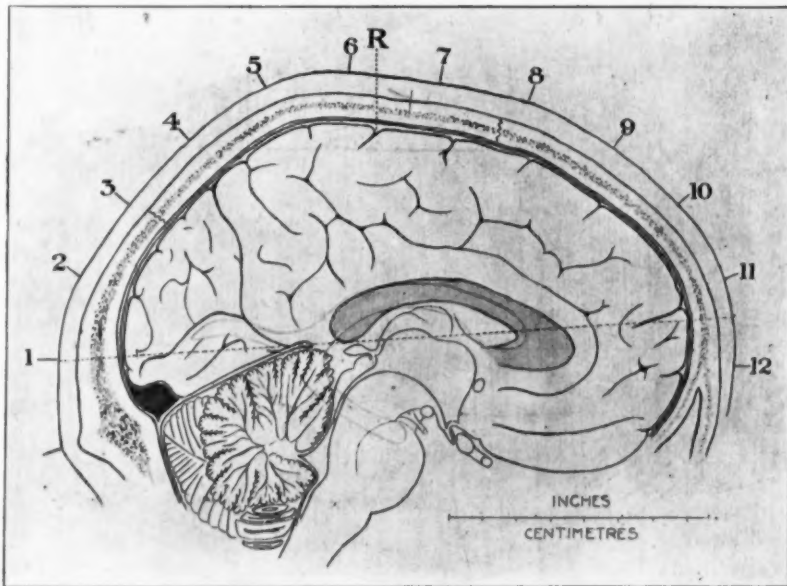


FIG. 2.

Mesial sagittal section of the head to show the relation of the calcarine fissure to the inion. The figures indicate the distance of each point from the inion in inches, measured on the surface of the scalp.

There is, however, enough evidence to show that the fibres in the radiations are to some extent arranged according to their destination in the area striata. Otherwise many published records would be unintelligible.

The injuries in our cases include penetrating and perforating wounds of the cranium by rifle bullets, by shell fragments and shrapnel, as well as local concussions and depressed fractures.

The portions of the brain injured can be approximately determined when there is a depressed fracture or a penetrating wound of the skull by noting its relation to certain fixed points, as the inion, supplemented by a stereoscopic X-ray examination. If the relations of the calcarine fissure and of the area striata to this fixed point are approximately constant, as we have found them to be (fig. 2), the position of the cerebral injury can be often fixed within relatively close limits, and if

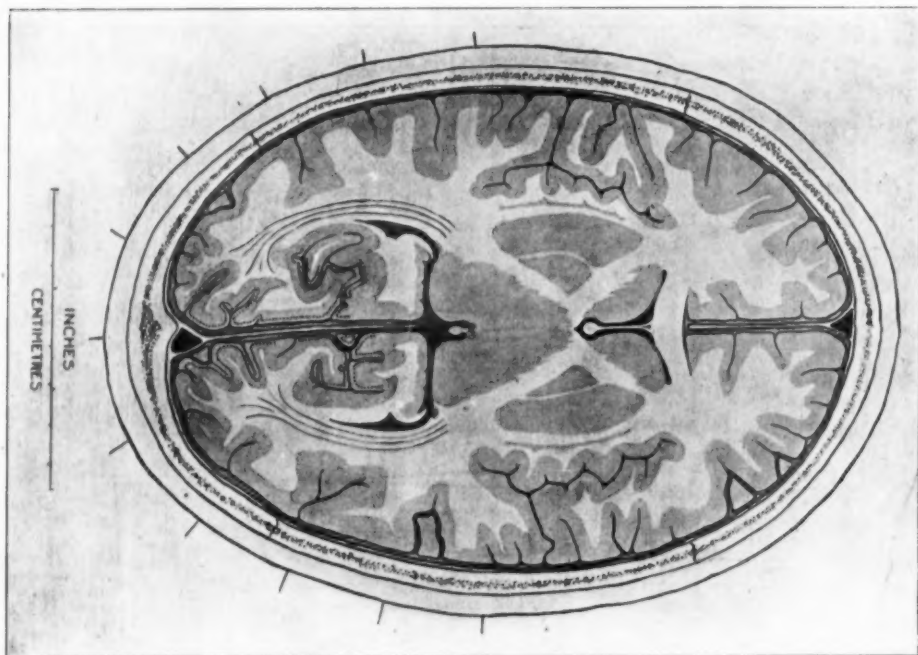


FIG. 3.

A horizontal section through the skull, passing 1 in. above the inion and 2 in. above the nasion, in the plane represented by the broken line in fig. 2, to show the relations of the area striata. This is indicated by a broken line in the cortex. The marks on the surface of the scalp represent inches measured in this plane from the middle line posteriorly.

the anatomical relations of the whole of the visual cortex, as seen in both sagittal and horizontal sections (fig. 3), are also known, then in the case of traversing bullets and fragments of shell, the entrance and exit wounds of which can be measured from a fixed point, the track of the missile can be calculated, since experience has shown

that bullets usually take a straight course between the entrance and exit wounds. It must be remembered, however, that the amount of destruction produced by such a missile may vary much. In many of the cases we have been also able to determine the approximate site and extent of the cerebral injury during operation, and in a few cases we have been able also to verify it by post-mortem examination. But though the exact localization of the lesion may appear to be indefinite, we believe that the consensus of evidence will be sufficiently strong to justify certain positive conclusions.

QUADRANTIC DEFECT IN THE VISUAL FIELDS.

We have been able to observe a certain number of cases of so-called quadrant hemianopia, and will first deal with them, selecting those that throw most light on the question of the cortical representation of the upper or of the lower quadrants of the retina:—

Case 1.—Lieutenant C. was wounded February 26, 1915, by a fragment of shell. He was unconscious for a short time, and afterwards had much headache, and found his sight was affected. He was operated upon next day in a casualty clearing station, when pieces of depressed bone were removed, and a week later he was sent to the base.

Wound.—There was then an oval open wound surrounded by a flap incision; its centre was $3\frac{1}{2}$ in. above and 1 in. to the right of theinion, and from here a track, closed at its lower end, but open for about $\frac{3}{4}$ in., passed horizontally forward into the brain. He had then loss of vision in the lower left quadrants which came to within 5° of the fixation point. He presented no other abnormal symptoms (fig. 4). His wound healed rapidly, and he was evacuated to England. It is obvious that in this case the wound, which was of considerable size, lay above the right calcarine fissure.

Case 2.—Staff-Serjeant M. was wounded by a piece of shell casing on May 23, 1915. He was unconscious for a short time, and later had very severe headache and vomiting. He also found his vision seriously affected at first. When he arrived at the base next day there was a large wound, the centre of which lay $2\frac{1}{2}$ in. above theinion and $\frac{1}{4}$ in. to the right of the middle line. An X-ray plate showed fragments of bone and a small piece of metal driven into the brain, directly forwards, to a considerable depth; these were removed with some septic and disintegrated brain, and the track drained. He made an uninterrupted recovery and was transferred to England about three weeks later. While under observation he had a complete loss of vision in the lower left quadrants and slight limitation in the periphery of the left upper quadrants (fig. 5). In this case, too, in which a considerable amount of brain was probably damaged, the lesion lay above the level of the right calcarine fissure.

Case 3.—Lance-Corporal M. was wounded by a bullet on March 24, 1915. He was not unconscious, but had severe frontal headache and vomiting³ for some days. He was completely blind at first, but on the fifth day was able to recognize light to the right of the fixation point.

The wound, which was $1\frac{1}{2}$ in. above the inion, extended across the middle line; it had been operated upon in a casualty clearing station, and depressed fragments of bone were removed. An X-ray examination later showed a defect in the skull, chiefly to the right, and a linear fracture running forward on the

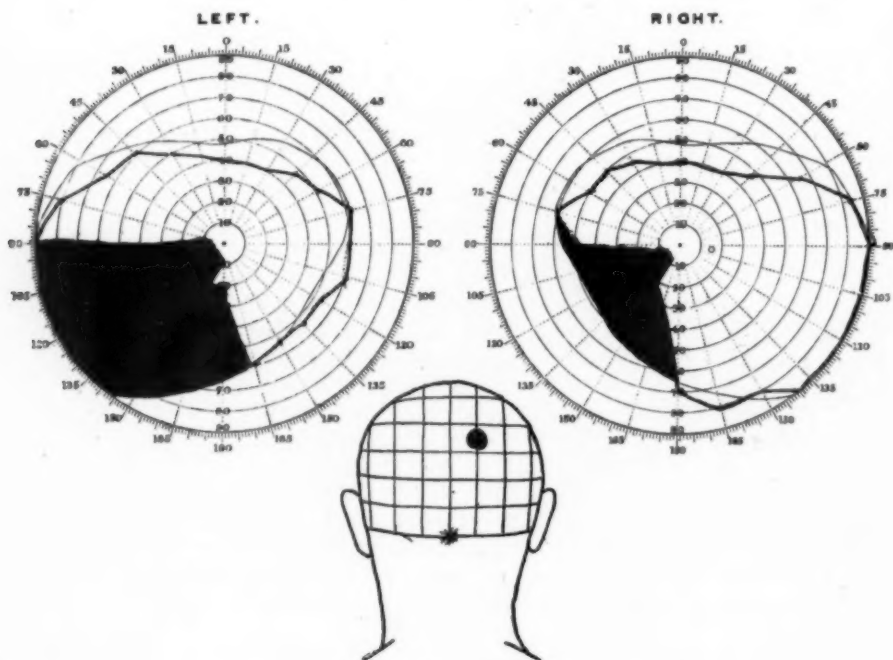


FIG. 4 (Case 1).

In this and the following figures the position of the wound is represented approximately on a diagram of the back of the head. The cross lines on this represent the distance of the plane in inches above the inion, the vertical the distance from the middle line of the skull.

right side. When seen at the base five days later he had distinct vision only immediately to the right of the fixation point, but on the seventh day had an almost full field in the right upper quadrant, but could recognize colours only by central vision. On the fifteenth day after the infliction of the wound there was some further recovery in the lower right quadrant near the fixation point,

and the colour fields in the seeing portion of the fields was almost full (fig. 6); on the left the blindness extended up to the fixation point. In this case the lesion lay immediately above the posterior end of the calcarine fissures; it evidently produced considerable destruction in the right hemisphere, and probably affected only the upper portion of the calcarine cortex on the left.

Case 4.—Private J. was wounded about December 1, 1915, by a bullet. He was unconscious for some days, and, on regaining consciousness, found he was quite blind, but on the next day he could see to the right side. There was no paralysis, sensory disturbance, or reflex changes.

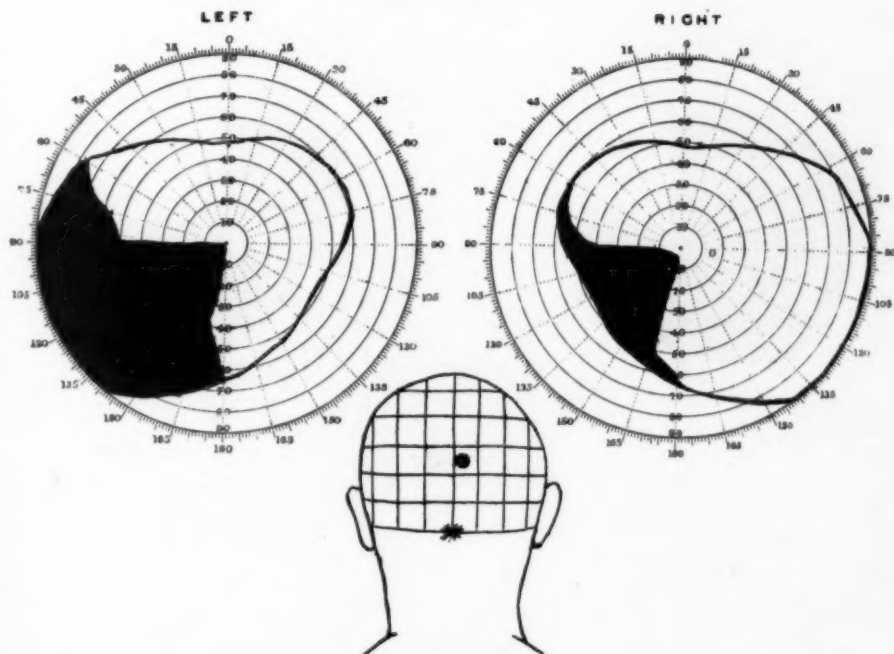


FIG. 5 (Case 2).

Wound.—The bullet entered in the right lower parietal region $2\frac{1}{2}$ in. above and $1\frac{3}{4}$ in. behind the upper margin of the attachment of the right auricle, and made its exit 2 in. above the inion and $1\frac{3}{4}$ in. to its left. On his arrival at the base, probably about a week later, small flaps were turned down around both wounds and some depressed fragments of bone removed from the entrance. He made an uneventful recovery. About three weeks after the infliction of the wound his visual fields were carefully examined with test objects 4 mm. in diameter, and it was found that his vision was limited to the left upper

quadrants. The blindness approached to within 3° of the fixation point (fig. 7) In this case the bullet probably entered in the right angular gyrus, injured the upper part of the right optic radiations beneath it, and, assuming it had gone straight through the skull, it would have passed through the upper part of the right area striata and then entered the left hemisphere through the middle of the area striata, probably near the parieto-occipital fissure.

We have so far seen only one case of horizontal hemianopia in which the patient's state permitted a careful perimetric examination, but even in this patient there was in addition a superior paracentral scotoma.

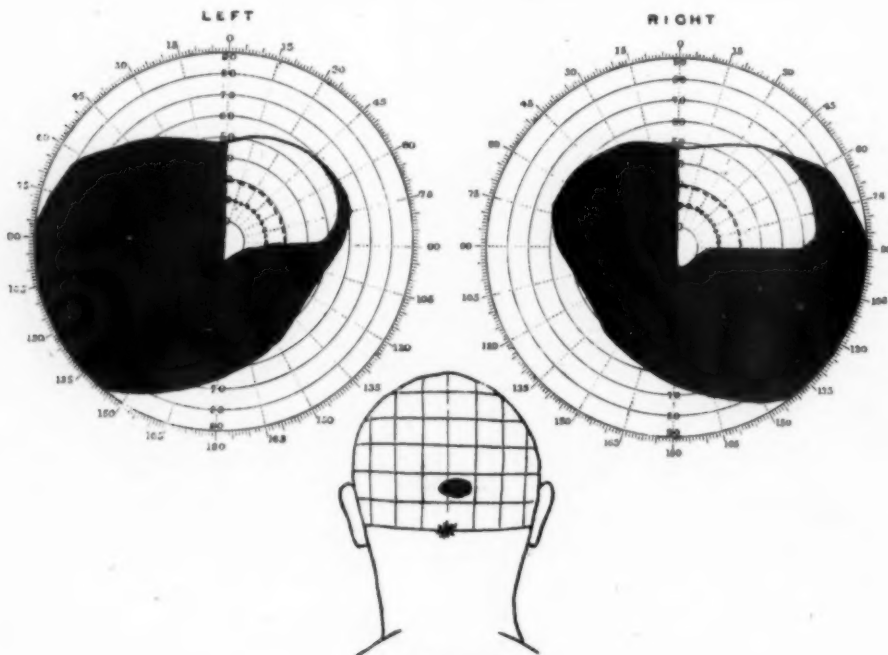


FIG. 6 (Case 3).

Case 5.—Private P. was wounded by a rifle bullet on September 25, 1915. He was unconscious for about twelve hours, and thereafter had severe headache and found his sight misty. He arrived at the base three days later.

Wound.—The entrance was a small puncture 3 in. to the left of the middle point of the vertex, the exit being 1 in. above and $1\frac{1}{2}$ in. to the right of the inion. An X-ray examination showed a small entry wound through the skull with a few small fragments of bone driven in, and a larger blown-out exit wound to the right of and about $1\frac{1}{2}$ in. above the inion. There was then some general weakness of his right arm and of this side of his face, but his legs

were equally strong, and the only changes in his reflexes were absence of the right arm-jerks and diminution of the right abdominal reflex. There was also considerable sensory disturbance of the cerebral type in his right upper limb, the appreciation of position, passive movement and form, and the discrimination of compass points, being lost. The motor symptoms disappeared almost entirely while he was under observation, but some sensory disturbance persisted. His headache was relieved by lumbar puncture and did not recur. His wounds healed rapidly. When he was

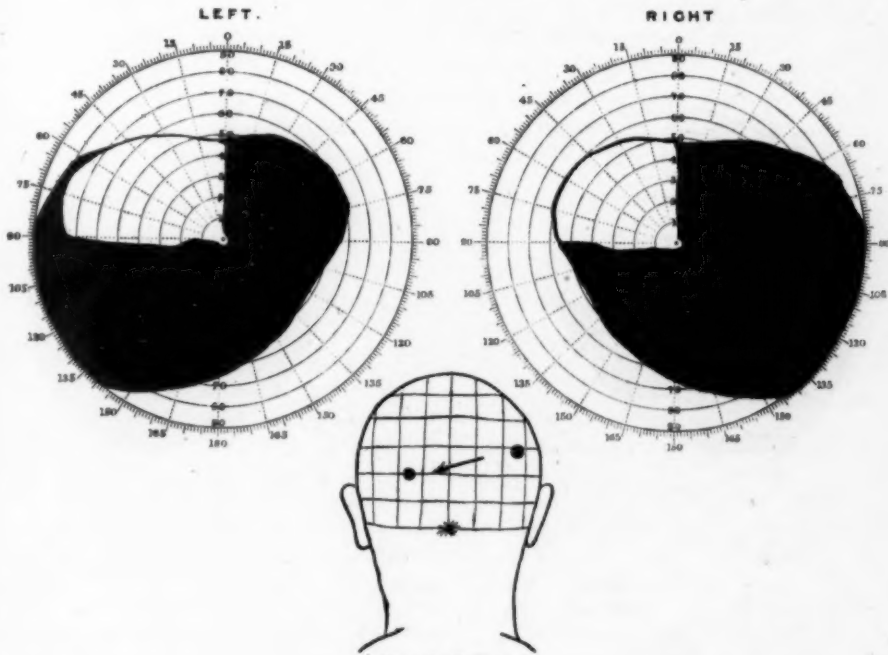


FIG. 7 (Case 4).

first seen, four days after the infliction of the wound, central vision was defective, and he was unable to see below a horizontal line through his fixation point. On October 11, however, central vision was $\frac{5}{6}$ in each eye, and he easily read Jaeger 1, but there was obviously a scotoma to the left of and above the fixation point. A few days later, when he was able to leave his bed, his visual fields were taken carefully and a complete lower horizontal hemianopia, almost limited by a horizontal line through the fixation point was discovered. There was an absolute left superior paracentral scotoma which came almost up to the fixation point (*see below*) and extended out to 10° (fig. 8). Colour vision was normal at the fixation point and in the superior quadrants,

except in the position of the scotoma, where its loss was coterminous with that for white. He remained under observation for five weeks after the infliction of the wound, and during the last three weeks of this period the visual disturbances remained stationary. In this case the bullet probably entered the left posterior central gyrus at about the level of the superior genu of the fissure of Rolando, passed obliquely backwards, downwards and inwards through this hemisphere in the neighbourhood or through the upper portion of the sagittal strata, and, from the position of the exit wound, probably cut through the upper part of the right calcarine area and through its posterior extremity at the pole of the hemisphere.

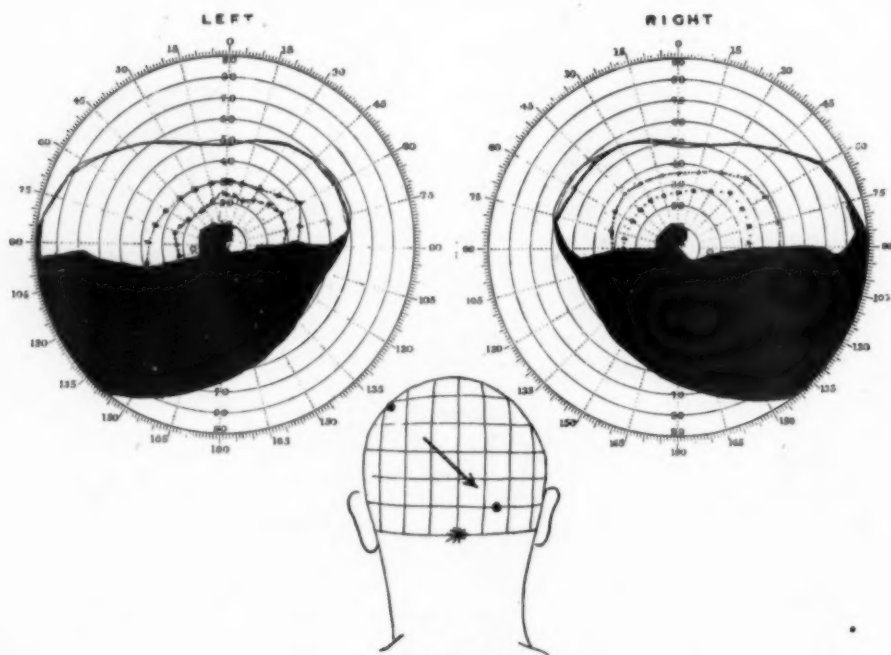


FIG. 8 (Case 5).

It will be noticed that in those cases in which one quadrant only was affected it was always the lower quadrant of the visual field, that is, the sector which corresponds to the opposite superior quadrant of the retina, which was blind; this was so in all the cases in which we have observed complete or partial quadrantic blindness. Further, in all cases the position of the injury or the course of the missile made it probable that the brain was damaged chiefly above the level of the calcarine

fissure, and our cases consequently support Henschen's view, for which there is now much evidence, that the upper halves of the retinæ are represented in the upper lips of the calcarine fissures. Inouye's observations on cases injured in the Russo-Japanese War point to the same conclusion.

The rarity of superior quadrantic hemianopia in gunshot injuries of the occipital region is striking; it is probably due to the fact that through-and-through or penetrating wounds in the inferior part of the area striata are liable to produce lesions in the cerebellum, which are notoriously very fatal. The fact that only the upper quadrants on one side escaped in Cases 3 and 4, in which the lesion on the opposite side probably lay above the level of the calcarine fissure, is evidence that the inferior portions of the retina are represented in the inferior parts of the area striatæ.

CENTRAL SCOTOMATA.

We have had the opportunity of seeing a considerable number of central and paracentral scotomata, and will describe here those cases which bear most definitely on the vexed question of the cortical representation of the macula.

Case 6.—Private F. was wounded by a bullet on July 11, 1915. He was unconscious probably for two days, and later had severe headache and found his sight seriously affected.

Wound.—There was a glancing wound across the middle line just above the inion, the entrance being on the right side and slightly above it, the exit on the left and immediately below its level. An X-ray stereoscopic examination showed a grooving of the outer table of the skull at the upper margin of the inion and the depression of a large flake of the inner table on to the poles of both occipital lobes. He was first seen four days after the injury; vision was then much disturbed, but it was not possible to take his visual fields by the perimeter till a week later. Then a large absolute central scotoma, which extended all round the fixation point to about 15° , except below, where it came within the 10° circle, was found. In this area there was complete blindness to white and to colours, the test objects being 2 mm. square. The peripheral fields for both white and colours were unrestricted. (In this and in the following cases, in which central vision was affected, fixation was obtained by placing the tip of the patient's forefinger on the fixation point of the perimeter and making him gaze at it.) He remained under observation for three weeks after the injury; towards the end of this period vision improved to some extent, so that only in the upper left quadrants was there complete blindness, while over the rest of the scotomatous area there was some, though indistinct, vision

(fig. 9). In this case it was only the tips of the occipital poles which were probably injured; they must have been bruised by the depressed portion of the inner table of the skull.

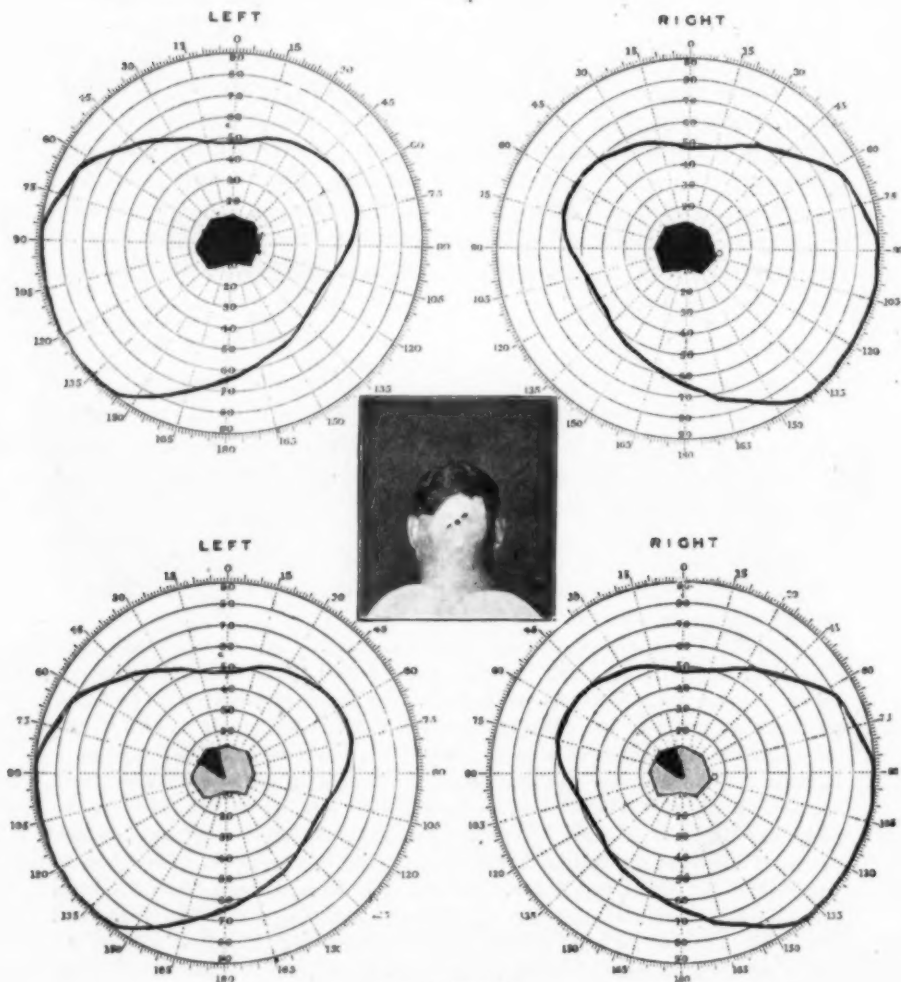


FIG. 9 (Case 6).

Case 7.—Private R. arrived at a base hospital on March 7, 1915. He had been unconscious for some days and could give no history of his injury or of an operation having been done at a casualty clearing station.

The entrance wound was $\frac{1}{2}$ in. to the left and $\frac{1}{2}$ in. above the level of theinion, while the exit was 2 in. to its right and 1 in. above its level. There was a small bone defect beneath each wound, through which tense cerebral herniæ protruded. That under the entrance wound did not pulsate. When admitted he had much headache and papilloedema with swelling of 1.5D. to 2D. The right eye was astigmatic and had been amblyopic since infancy, and he had a slight convergent squint. Apart from the visual disturbance there were no other symptoms of cerebral injury. His visual fields, taken about ten days after the infliction of the wound, showed a large central scotoma in each eye, which was somewhat more extensive in the right than in the left, and in the amblyopic eye there was also some peripheral contraction, which was probably due to want of attention (fig. 10). The scotomata were practically

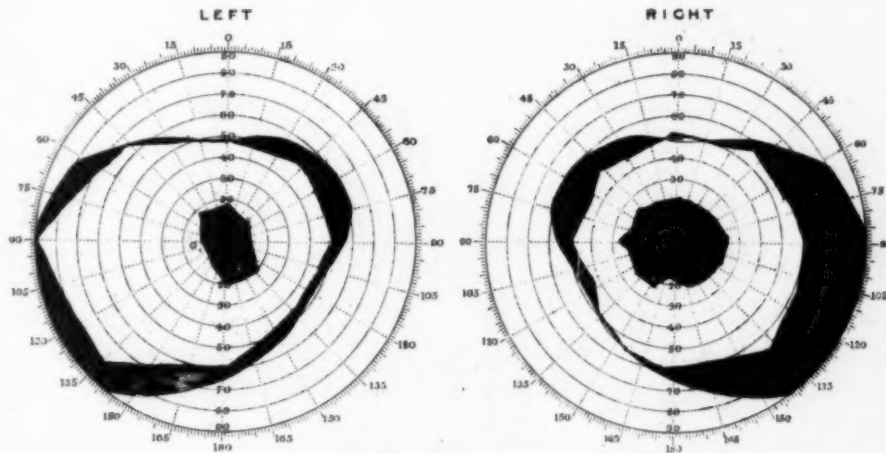


FIG. 10 (Case 7).

unaltered when his fields were taken again a few days later and remained so till he was transferred to England. In this case the wounds were due to a bullet which had passed horizontally across the occipital poles at about the level of the posterior ends of the calcarine fissures, and which must have consequently destroyed the posterior part of each area striata.

Case 8.—Private L. was wounded by shrapnel on March 11, 1915. He was completely blind for two days and had had much headache. There were several shrapnel wounds in the scalp, but the bone was injured only under a triangular wound immediately to the left of theinion. An X-ray examination showed a comminuted fracture in this region with depressed fragments of bone in the brain beneath it, and a linear fracture running upwards and outwards to the left parietal eminence. When first seen, four days after

the injury, he had complete right hemianopia and some defect of central vision. When his fields were taken eight days later, the peripheral fields were full, but a right absolute paracentral scotoma was discovered which extended outwards to about 20° and inwards to the fixation point. This was surrounded by a partial scotoma which invaded the left halves of the fields to the extent of about 10° round the fixation point. At this time he was unable to read even large letters. Both the complete and the partial scotomata gradually decreased in size, and about three weeks after the infliction of the wound there was only a small absolute paracentral scotoma in the right lower quadrant which was surrounded by an area of defective vision. The latter extended to about 5°

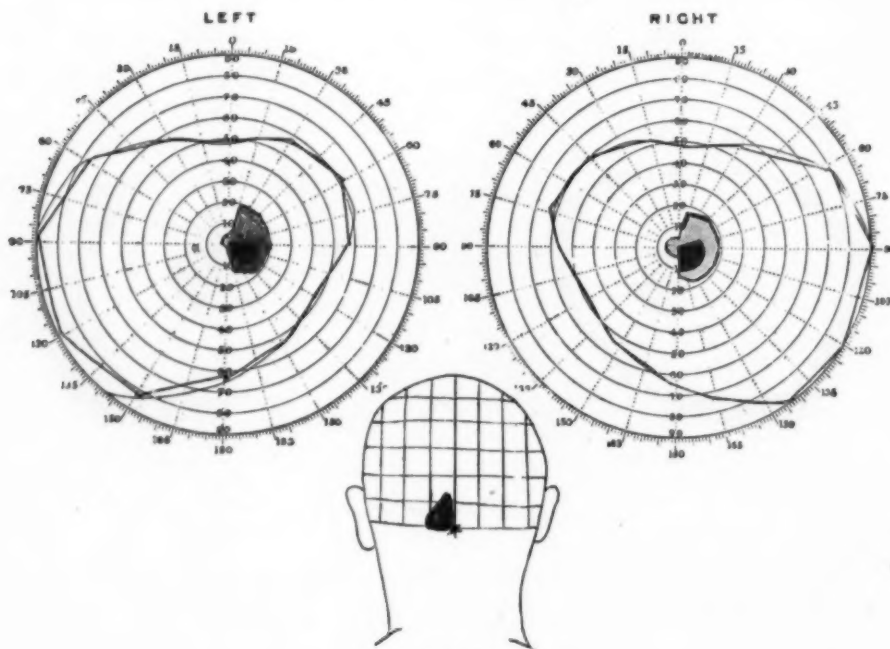


FIG. 11 (Case 8).

to the left of the fixation point (fig. 11). At this time he was able to read only the largest letters. In this case the main injury must have been at the tip of the left occipital pole, but the right occipital pole was probably also damaged to a slight extent.

Case 9.—Lieutenant T. was wounded on December 20, 1914, by a bullet. He was unconscious for a time, and on coming round found he was quite blind, and had a sensation of positive blackness in front of his eyes. He first noticed, about four days later, the white aprons of the sisters passing by his bed.

Wound.—The entrance lay about 1 in. above the level of the inion and 1 in. to the left. The exit was $2\frac{1}{2}$ in. above the inion and 2 in. to the right of the middle line. An X-ray plate showed a depressed fracture of the skull and fragments lying in and on the brain. He was operated on by Mr. Hugh Lett on December 23. A large horseshoe flap, which included the

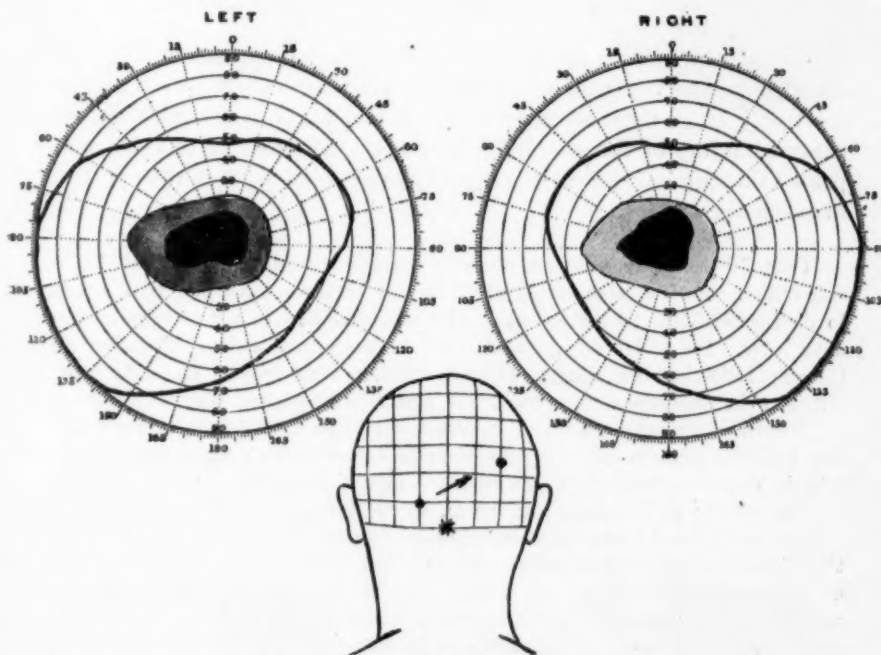


FIG. 12 (Case 9).

two wounds, was turned down. Brain matter at once escaped from the entrance. The openings in the bone on both sides were enlarged and the intervening bridge of skull removed. The dura had been torn from one opening to the other, and the damaged brain bulged through it after irregular fragments had been removed from both occipital lobes. The patient made an uninterrupted recovery. The visual fields were taken eleven days after injury; there was then a large absolute central scotoma extending about 10° on the right of the fixation

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point and to nearly 20° on its left. This was surrounded by a zone of partial vision (fig. 12). From this time his sight improved considerably till he was discharged twenty-six days after the injury. There was then very imperfect central vision, with an absolute paracentral scotoma below the fixation point, which was surrounded by an area of incomplete vision. He was now able to recognize large letters. The peripheral limits of the visual fields were unrestricted. In this case the course of the missile and observation of the injury at the time of operation make it certain that the tip of each occipital lobe was seriously damaged.

HOMONYMOUS HEMIANOPIA WITH CENTRAL SCOTOMA.

Another interesting group of cases that we have observed has been distinguished by the presence of homonymous hemianopia with a central scotoma. In all these the missile, which had an oblique course, caused extensive injury of the occipital lobe or optic radiations of one side, and passed through or near to the tip of the occipital pole of the opposite hemisphere.

Case 10.—Private G. was probably injured on March 26, 1915, by a rifle bullet. He was completely blind for two or three days, and then began to distinguish light to the left side. He was admitted to a base hospital five days later. He had been operated upon in the casualty clearing station on the day following the injury. He was at first very drowsy and had some difficulty in understanding speech, but this rapidly cleared up.

Wound.—The entrance was 2 in. above and $1\frac{1}{2}$ in. to the right of theinion. The exit was represented by a large circular defect in the scalp about $1\frac{1}{2}$ in. to 2 in. in diameter, through which softened and disintegrated brain protruded. Its centre was $2\frac{1}{2}$ in. above and $1\frac{1}{2}$ in. to the left of theinion. An X-ray photograph showed a large bone defect under the exit wound, which reached to the middle line and extended almost down to theinion. There was no paralysis or sensory disturbance, and his reflexes were normal. At first he had vision only in the left upper quadrants, but later vision returned in the whole periphery of the left visual field, though a large central scotoma extending to about 20° to the left of the fixation point persisted till he was transferred to England, a month after the infliction of the wound (fig. 13). In this case there can be no doubt that the posterior part of the optic radiations of the left hemisphere, and probably a considerable part of the calcarine cortex, were destroyed by the missile and by the secondary changes involved in the hernial formation, while on the right side the bullet merely passed close to the posterior extremity of the occipital lobe.

Case 11.—Private M. was wounded by a rifle bullet on September 25, 1915. He was blind for the first two days, and had a sensation of blackness

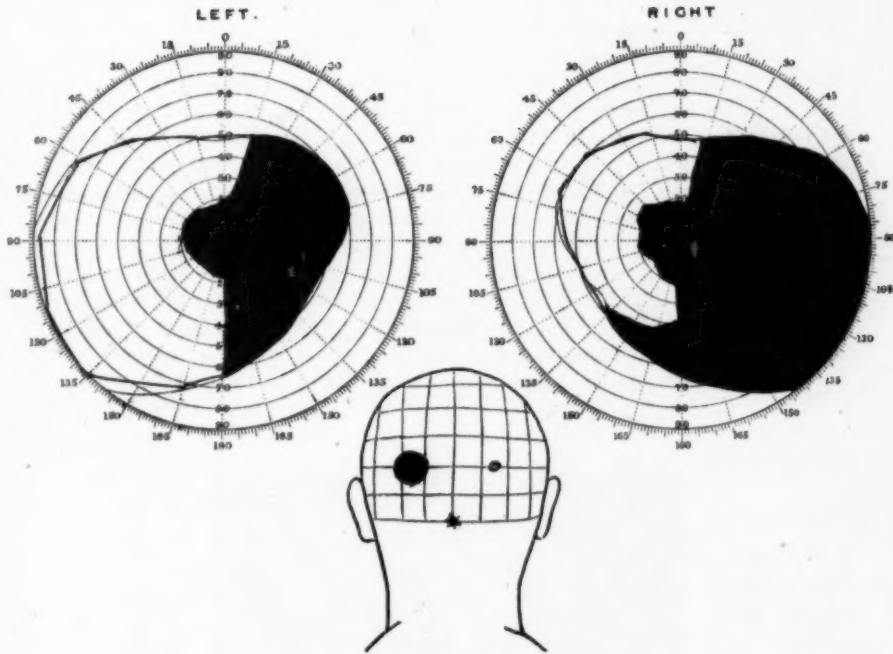


FIG. 13 (Case 10).

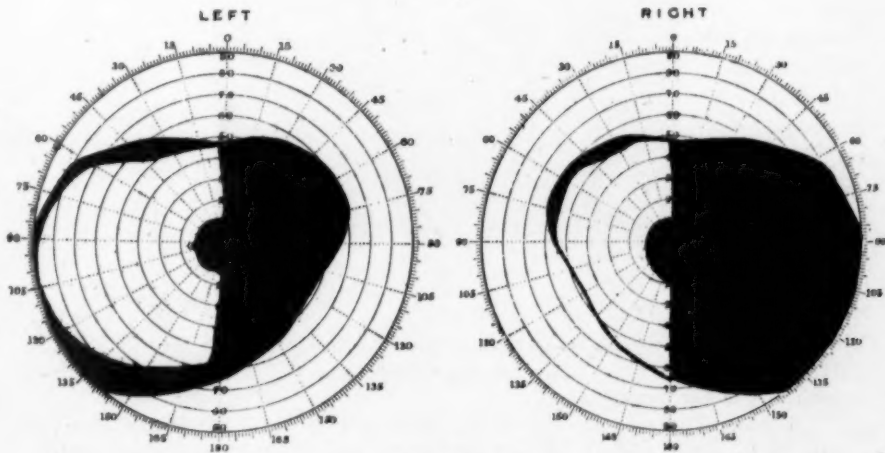


FIG. 14 (Case 11).

in front of his eyes. He reached the base three days later, and then presented no abnormal symptoms apart from visual disturbance.

Wound.—The entrance was 2 in. above and 3 in. to the left of the inion. The exit was 2 in. to the right and 2 in. above the inion. An X-ray photograph showed under the entrance wound a defect in the skull at the lambdoid suture, with many fragments of bone driven into the brain, while the exit was represented by a blown-out wound 1 in. to the right of and above the inion. The visual fields, taken three weeks after injury, show complete right-sided homonymous hemianopia and a central scotoma which extended to just beyond 10° to the left of the fixation point (fig. 14). His vision remained in this state till he was transferred to England. In this patient the injuries were similar to those in Case 10. The left optic radiations must have been injured by the depressed fragments of bone and by softening under the wound of entry, while owing to the oblique course of the missile it is probable that only the posterior extremity of the hemisphere was damaged on the right side, and chiefly on its mesial surface.

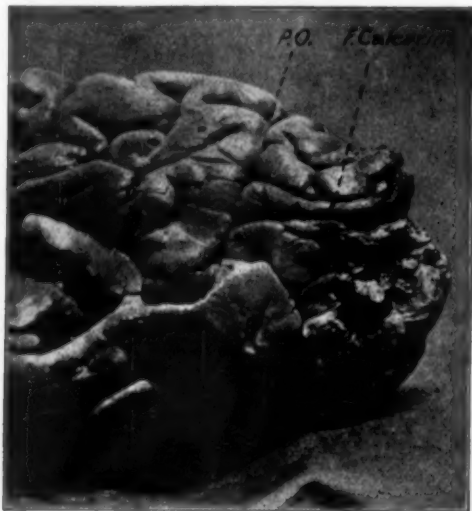


FIG. 15 (Case 12).

Case 12.—Private M. was admitted to a base hospital on January 13, 1916. He was then quite blind, but a few days later could recognize light in the left periphery of his visual fields.

Wound.—The entrance was $1\frac{1}{2}$ in. above and $3\frac{1}{2}$ in. to left to the inion; exit $1\frac{1}{2}$ in. above and $1\frac{1}{2}$ in. to right of the inion. An X-ray photograph showed fragments of bone driven into the brain beneath the entrance wound.

There was no paralysis, sensory or speech disturbances, nor alteration in his reflexes. The depressed fragments of bone with some disintegrated brain were later removed from the wound of entrance, but owing to the spread of infection to the ventricle and the occurrence of meningitis, he died five weeks after the infliction of the wound. The visual loss was so extensive that it was never possible to take his visual field with a perimeter, but during the whole time he was under observation he had complete right homonymous hemianopia with an absolute central scotoma, which extended for about 20° to 30° to the left of the fixation point. At the autopsy a large septic wound was found in the left occipital lobe, which involved all the structures lateral to the ventricle, including the optic radiations. The tip of the right occipital lobe protruded as a small hernia through the wound of exit, and on this side the bullet had, in passing, destroyed about the posterior third of the calcarine cortex (fig. 15). The brain was not infected on this side, and the rest of the area striata was undamaged.

In these seven cases (Cases 6 to 12), there were absolute central scotomata of various sizes. In the first four peripheral vision was unaffected, but in Cases 10, 11, and 12 the central scotomata were associated with homonymous hemianopia.

In those cases in which there was a pure central scotoma the lesion was, as far as it is possible to judge from the course of the missile, from the evidence afforded by radiographic examination, and from the conditions found at operation, limited to the posterior ends of the occipital lobes at or about the level of the posterior extremities of the calcarine fissures. Consequently it is probable that only the hindermost ends of the area striata were damaged. This was obviously so in Cases 7 and 9, while in Case 6 the depression of the inner table must have bruised and probably produced softening of the occipital poles. In Case 8 the cranial injury, it is true, was unilateral, but it is the common experience that such depressed fractures due to shrapnel or fragments of shell-casing often produce relatively diffuse damage; further, in this case the visual symptoms diminished rapidly, so that when he was last examined the absolute scotoma was only paracentral; its position in the right halves of the visual fields corresponded with complete functional abeyance of a portion of the left area striata, and it was over the posterior part of this that the lesion actually lay.

These observations, associated with the fact that among over 2,000 cases of head injury we have never seen a central scotoma when a direct injury of the occipital poles could be excluded, afford strong evidence that central vision is represented on either the mesial or the lateral surface of the posterior poles of the occipital lobes, or

on both. Further, our observations conform to the generally accepted view that the visual area corresponds with, or at least includes, the area striata.

Those cases (10, 11, and 12) in which there was a homonymous hemianopia in addition to a central scotoma also conform to or support this view. In Cases 10 and 11 the hemianopia was probably due to the extensive lesions on the lateral surfaces of the occipital lobes on one side, which must have involved the optic radiations, while, owing to its oblique course, the missile can have injured directly only the posterior pole of the opposite hemisphere. In Case 12, in fact, in which the visual fields were, as far as could be ascertained by rough methods of examination, very similar to those of the two preceding cases, the optic radiations and the greater part of the calcarine cortex were destroyed on the left side, while in the right hemisphere the destruction involved only the posterior part of the area striata; and this lesion corresponded with loss of central, but with intact peripheral vision in the left halves of the visual fields.

It is true that in all such lesions afferent sensory tracts must be damaged in addition to the cortex, but as the fibres of the optic radiations destined for the anterior extremity of the area striata do not extend farther posteriorly than the horn of the ventricle (fig. 3), it can be assumed that when the lesion is limited to the occipital pole centripetal fibres to this region only are involved.

The evidence so far consequently points to representation of the macula at the posterior pole of the hemisphere. This conclusion is strongly supported by the next group of visual disturbances with which we shall deal.

PARACENTRAL SCOTOMATA.

We have seen a considerable number of cases with paracentral scotomata—that is, isolated areas of blindness in the visual fields which lie to one side of the fixation point, but do not involve it.

Case 13.—Private B. was wounded by a piece of shell-casing on February 3, 1916. For an hour or so he had a "dim mist" in front of his eyes, but this gradually disappeared, and when he was admitted to the base hospital two days later he was unaware of any defect of vision.

Wound.—A small contused scalp wound, the centre of which was 1 in. above and $\frac{1}{4}$ in. to the left of the inion. An X-ray photograph showed a narrow crack in the outer table and a small depression of the inner table immediately

under the wound. The scalp wound was excised, but the skull was left untouched. When the visual fields were taken, twelve days after the infliction of the wound, the limits to those for white, red, and green were unrestricted, but there was a small absolute scotoma in the right inferior quadrant which reached to within 1° of the fixation point, and extended outwards to 10° (fig. 16). The areas over which perception of white and that of colours were

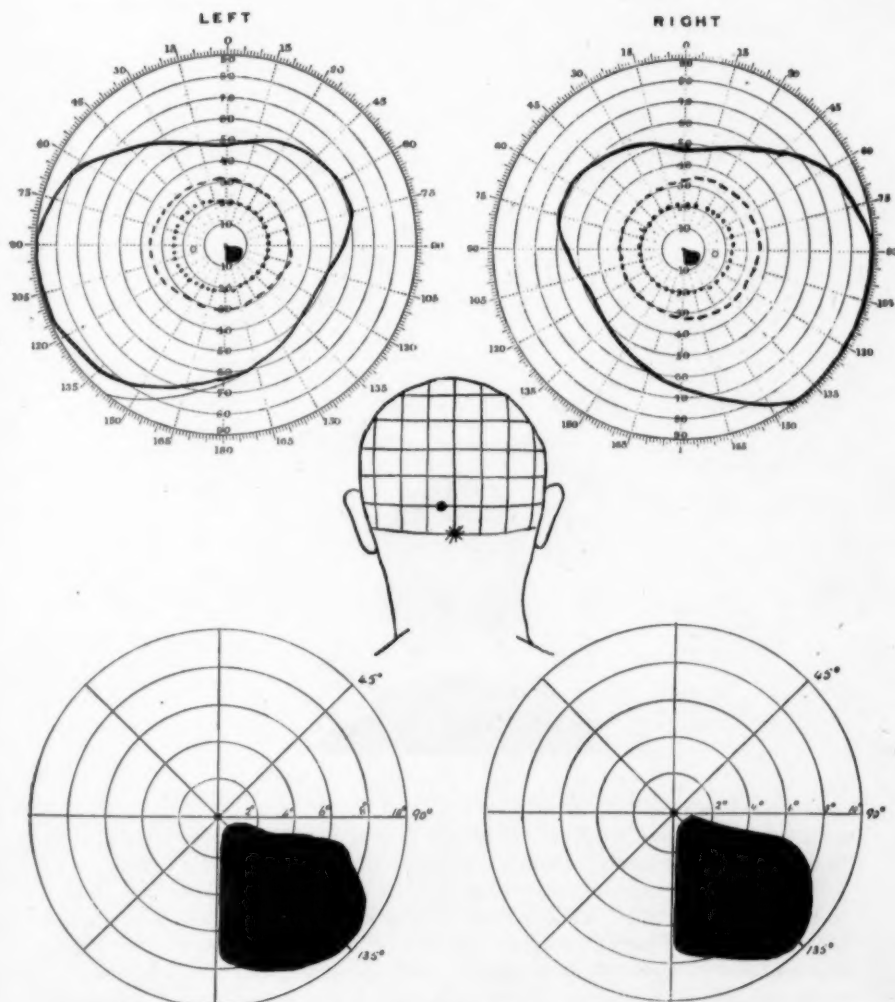


FIG. 16 (Case 13).

lost were identical. The position of the wound and the X-ray examination make it probable that in this case there was a contusion of the left occipital pole at the upper lip of the calcarine fissure.

Case 14.—Lieutenant H. was wounded on November 5, 1915, by a bullet. He noticed at first he could not see his feet, but his vision improved rapidly, and when he was first seen by us four days later, his only complaint was that

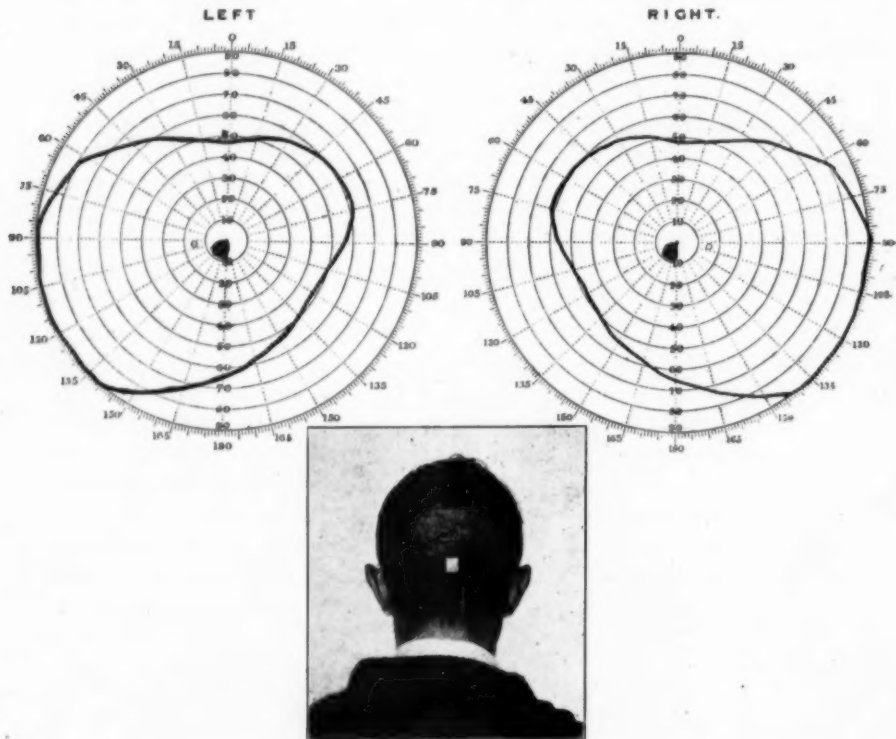


FIG. 17 (Case 14).

on attempting to read he could not see the four or five lines immediately below that which he was reading, though the rest of the page was visible to him.

Wound.—A tangential scalp wound with its entrance 1 in. above and $1\frac{1}{2}$ in. to the right of the inion, while the exit was in the middle line and $\frac{1}{2}$ in. above the inion. An X-ray photograph showed no definite changes in the skull. Examination of visual fields revealed a small absolute paracentral scotoma for white and colours which lay chiefly in the left lower quadrants; it extended up to the fixation point and downwards and outwards to about 9° (fig. 17).

This condition remained unaltered three weeks after the infliction of the wound. In this case the wound lay immediately above the posterior extremity of the upper lip of the right calcarine fissure.

Case 15.—Private A. was wounded on February 12, 1916, by a piece of shrapnel. He did not become unconscious, but immediately noticed his sight was affected. He was operated upon at a casualty clearing station on the following day, when a depressed fracture of the occipital bone was found

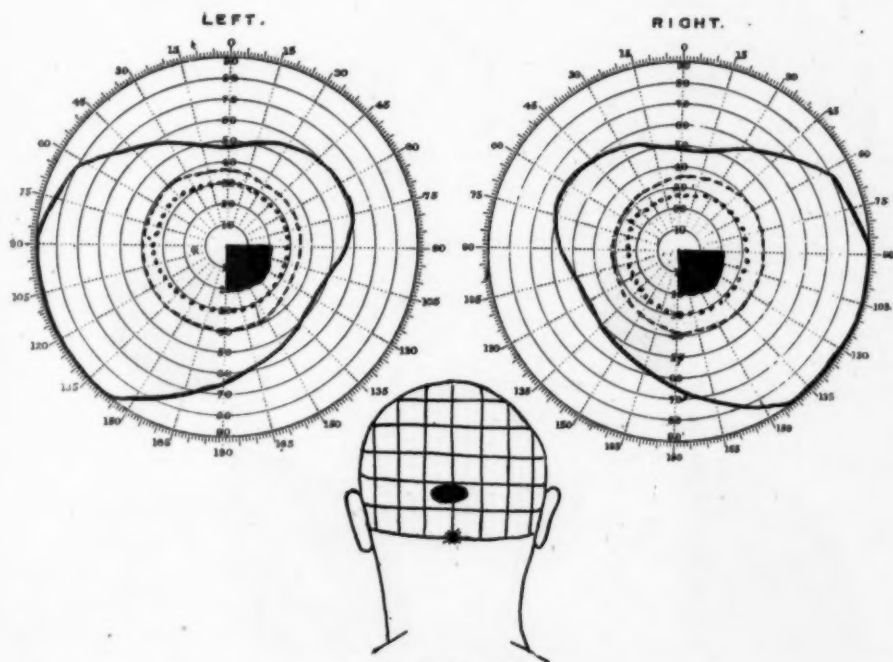


FIG. 18 (Case 15).

over the longitudinal sinus. The dura was probably undamaged. When admitted to the base five days later there was a transverse wound across the middle line $1\frac{1}{4}$ in. above the level of the inion, and an X-ray photograph revealed a defect in the skull midway between the inion and lambda in the middle line. Perimetric examination showed that the peripheral limits of white and colours were unrestricted, but there was an absolute scotoma for both white and colours in the right lower quadrants which extended from the fixation point to about 20° outwards and downwards (fig. 18). His central vision was $\frac{5}{8}$ in each eye.

Case 16.—Lieutenant F. was wounded March 13, 1915, by a bullet. His sight at once became indistinct. Two days later he was operated upon at a casualty clearing station.

Wound.—Entrance was $1\frac{1}{2}$ in. above and $2\frac{1}{2}$ in. to the left of the inion. The exit was in the middle line and 2 in. above the inion, and they were joined by a healed incision, beneath which a defect in the skull could be felt. Perimetric examination was first made a fortnight later. He then had a complete scotoma for white and colours in the right inferior quadrants which

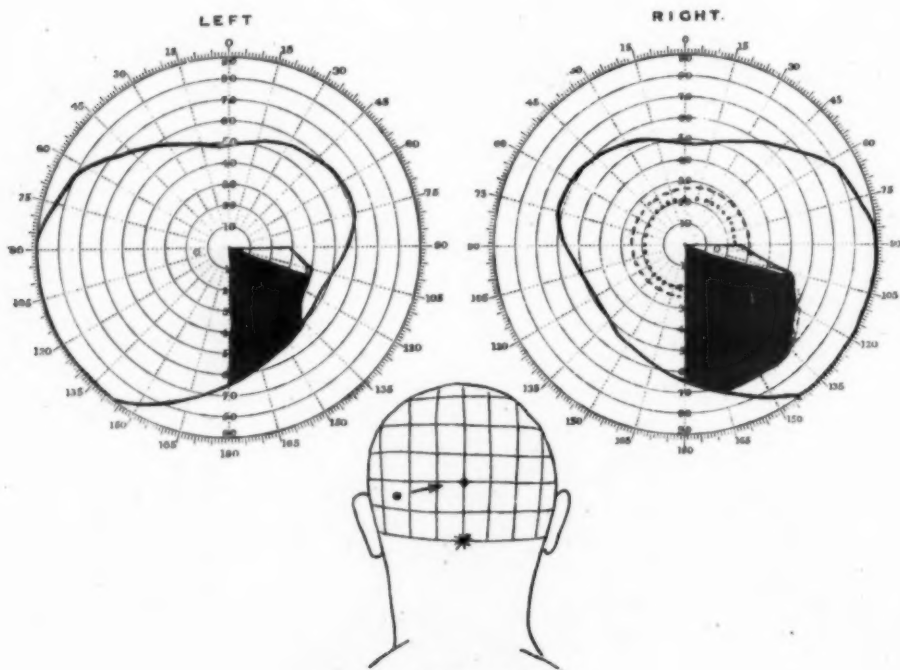


FIG. 19 (Case 16).

reached from the fixation point to the limit of the normal field immediately below; but to the right of this there was peripheral vision to both white and large areas of colour, which shows that the defect in the field was of the nature of a paracentral scotoma rather than a quadrantic loss (fig. 19). At the upper border of the scotoma the blindness was only partial from the horizontal line to 15° below it. In this case the extent of the damage was uncertain, but the wound lay over the upper and posterior portion of the left area striata.

Case 17.—Private C. was wounded by a rifle bullet on January 5, 1916. He was unconscious for two hours, and then found his sight much affected; it, however, rapidly improved.

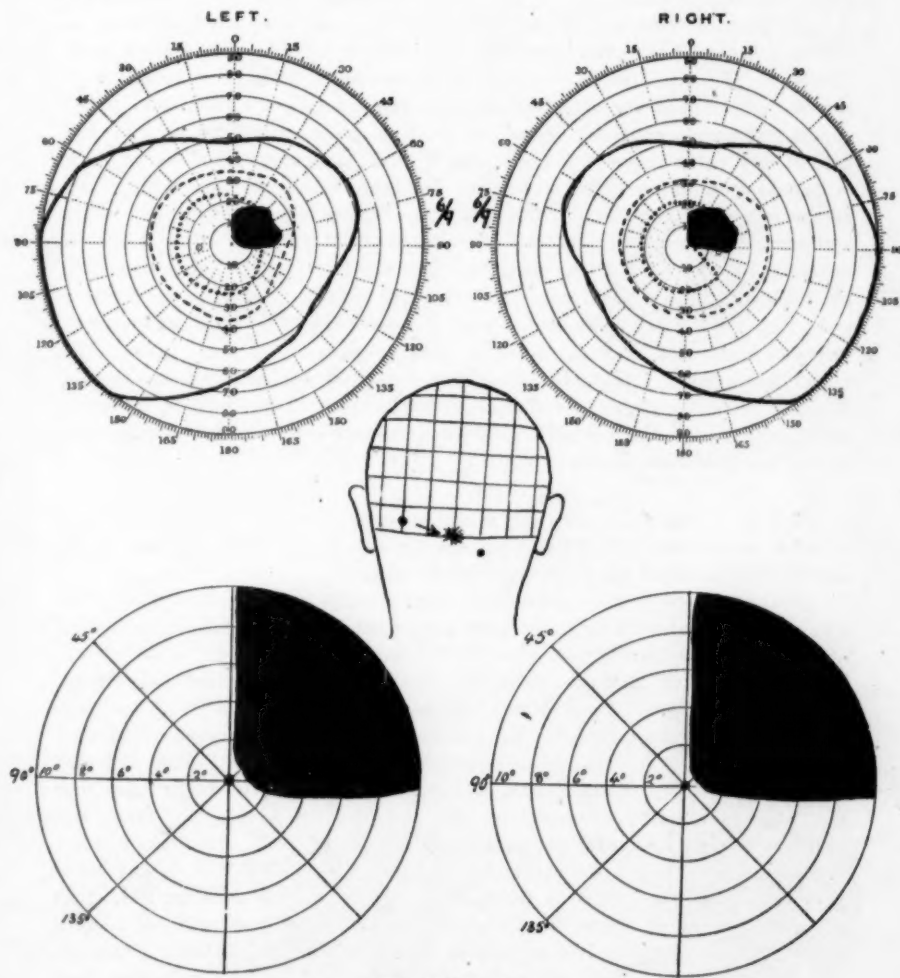


FIG. 20 (Case 17).

Wound.—The entrance was 2 in. to the left, $\frac{1}{2}$ in. above the inion; exit 1 in. to the right and slightly lower than the inion. An X-ray examination showed a medium-sized entry wound in the skull with depressed bone

beneath it, and an exit with out-driven bone immediately to the right of theinion. Two days after arrival at the base he was operated upon. A horse-shoe flap was turned down, and it was seen that the entrance lay just above the lateral sinus on the left side. The sinus was firmly thrombosed. A large flake of bone was removed from the brain, and several small pieces, as well as a firm clot from the upper margin of the cerebellum. He made a rapid recovery. His visual fields were first examined three weeks after infliction of the wound. There was an absolute paracentral scotoma in the right upper quadrants which extended from the fixation point outwards to 20° , and projected slightly below the horizontal. This was surrounded by an area of partial scotoma, in which white was dull and indistinct, and colours not recognized with certainty. The peripheral limits of the fields for white and red were unrestricted, but green objects of 10 mm. square could not be recognized to the right of the scotoma. The absolute scotoma, when measured by Bjerrum's screen, was found to extend up to less than 1° of the fixation point (fig. 20). His visual acuity was, however, $\frac{6}{6}$. The only complaint of vision he made was that in reading he had occasionally lost his place. His visual fields were repeatedly taken, but remained unaltered till he left for England six weeks after the infliction of the wound. There can be no doubt that in this case the occipital pole was injured chiefly below the extremity of the left calcarine fissure.

Case 18.—Lance-Corporal G. was wounded by a bullet as he was bending forward on January 26, 1916. He was unconscious for half an hour or so, and afterwards noted his sight was slightly affected.

Wound.—There was a long oblique wound which extended from the middle line of his skull about 2 in. above inion downwards and to the left for a distance of $2\frac{1}{2}$ in. On separating the edges of the wound a narrow gutter defect was found in the skull through which disorganized brain protruded. An X-ray examination showed a large flake of bone driven into the left occipital pole. Three days after the injury the depressed bone was removed and some softened brain washed away. His head wound healed rapidly. The bullet had also passed through the posterior part of his left shoulder. His fields were first carefully examined by the perimeter one month after the injury. There was found a large paracentral scotoma, which extended from the fixation point into the right halves of the fields to the extent of 20° to 30° , and in the lower portion of the left halves of the fields to the extent of 25° (fig. 21). The peripheral limits of vision for white were normal, but green and red objects 10 mm. in diameter were visible only to the left of the vertical line. Examination by Bjerrum's screen showed that the blindness extended to less than $\frac{1}{2}^{\circ}$ from the fixation point. His visual acuity was $\frac{6}{6}$ in each eye. His fields were taken repeatedly, but remained practically unaltered till he left for England six weeks after his injury. In this case the left occipital pole, both above and below the level of the calcarine fissure, must have been extensively damaged, while the right hemisphere was probably injured at the level of the

upper part of the wound by the in-driven flake of bone, which probably penetrated the falx cerebri. The exact extent of the lesion could not be accurately determined at the operation.

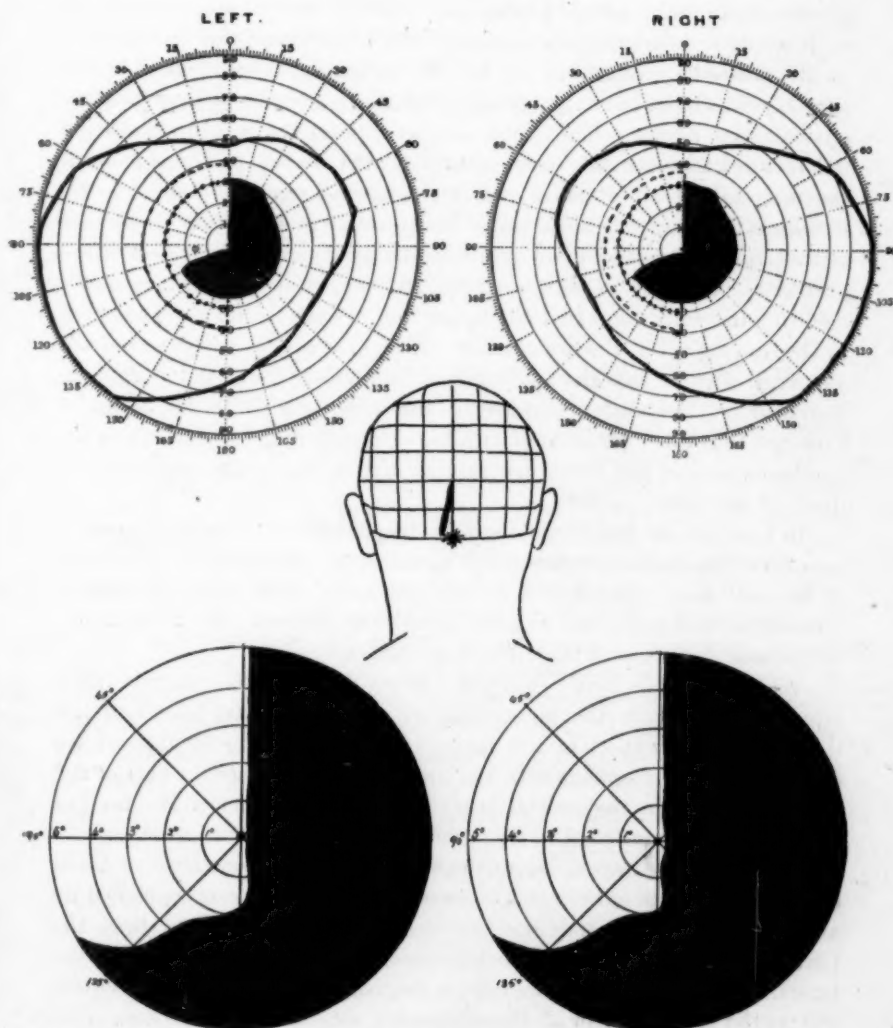


FIG. 21 (Case 18).

In these six cases the paracentral scotomata, which varied in size, appeared on ordinary perimetric examination to reach immediately to the fixation point, and on more careful investigation by a Bjerrum's screen they were found to come within at least 1° of it. If the fovea centralis subtends an angle of 4° to 6° (Starling), vision subserved by it would be consequently involved. In these cases the lesions were in all probability relatively superficial, except in Case 16, and in all they lay over or in the neighbourhood of the posterior extremity of the one calcarine fissure. They consequently support the conclusion already put forward that central vision is represented in the posterior parts of the occipital lobes. Further, it will be observed that in Cases 13 to 16, in which the paracentral scotoma was limited to the lower quadrants of the visual fields, the main lesion probably lay above the level of the posterior extremity of the calcarine fissure; this fact consequently confirms the conclusion that the upper half of each retina is represented in the dorsal part of the area striata. Case 17 is the only one we have yet seen with a superior paracentral scotoma; in it the brain was damaged on the under surface of the pole, and it is consequently evidence that those portions of the lower quadrants of the retina in the neighbourhood of the fovea are represented in the lower and posterior parts of the area striata.

In Case 18, in which we found a triquadrantic scotoma, the whole breadth of the posterior part of the visual cortex was probably destroyed on the left side. The defect in the left visual field, which, it will be noticed, did not reach the fixation point, was probably due to an injury of the mesial surface of the right hemisphere.

We can consequently conclude that not only is central vision represented towards the posterior part of the occipital lobe, but also that the upper portion of the retina in the immediate neighbourhood of the macula corresponds with the upper and most posterior part of the area striata, while the central portion of the retina below the level of the macula is represented in its lower and posterior part.

It is obviously impossible to draw definite conclusions from material such as ours, since our clinical observations have not been controlled by anatomical examinations, but we can suggest tentatively that the portion of the area striata which extends to the margin and on to the lateral surface of the occipital lobe is the cortical focus of central vision. For in the first place, in all the scotomata we have observed associated with injury to this region the blindness involved, or reached to within

1° of, the fixation point, though their sizes varied considerably; in other words, the fixation point or its immediate vicinity was that portion of the visual field which was constantly affected by posterior occipital lesions.

In the second place, in such cases as 6, 13, 14 and 16 the depression of bone upon, or the effects of concussion immediately over, the posterior aspect and the lateral surface of the occipital lobe produced a local disturbance of vision which extended up to or involved the fixation point.

Again, if the macula is represented on the posterior aspect of the hemisphere we should expect that wounds which penetrate the occipital lobe at the level of the extremity of the calcarine fissure would produce homonymous blindness reaching to the fixation point, and this we have actually found to be the rule. The following case illustrates it:—

Case 19.—Lieutenant C. was wounded by a bullet May 9, 1915. He was unconscious for some time and could give no history. When admitted to the base hospital three days later he was very dull and could recognize objects only to the right of the fixation point.

Wound.—There was a gaping gutter wound on the right side, parallel and close to the middle line, which extended from theinion $2\frac{1}{2}$ in. upwards. X-ray examination showed a linear defect in the skull and several pieces of bone driven deeply into the brain. An operation was performed the next day; a hole in the skull 2 in. by $\frac{1}{2}$ in. was found, the dura was lacerated for 1 in. above the inion and immediately to the right of the longitudinal sinus. Disintegrated brain protruded through it. Fragments of bone were removed from a long track, which passed directly forwards to a depth of about 2 in. There was considerable destruction of brain around the track. A perimetric examination was made two weeks after infliction of the wound; he had then complete left hemianopia extending right up to the fixation point (fig. 22).

But any such hypothesis on the cortical localization of central vision must be capable of explaining all generally accepted facts. The most important of these is the frequent escape of macular vision in homonymous hemianopia due to vascular occlusion. The explanation probably depends on the vascular distribution in the region of the occipital pole. According to Beevor's classical work on the distribution of the cerebral vessels, the occipital pole is usually supplied by the posterior cerebral artery, but the middle cerebral extends usually to

within $\frac{1}{2}$ in. to $\frac{1}{4}$ in. of it, and occasionally actually includes it (fig. 23). The occipital pole, and particularly its lateral aspect, is consequently a watershed area between these two large vessels; it may under normal conditions draw blood from both, and if one is blocked the other may suffice to maintain its nutrition. It is, in fact, well known that the softening due to occlusion of any cortical artery is rarely as extensive as its anatomical distribution. If this view is correct the presence or absence of macular escape in hemianopia due to vascular lesions may

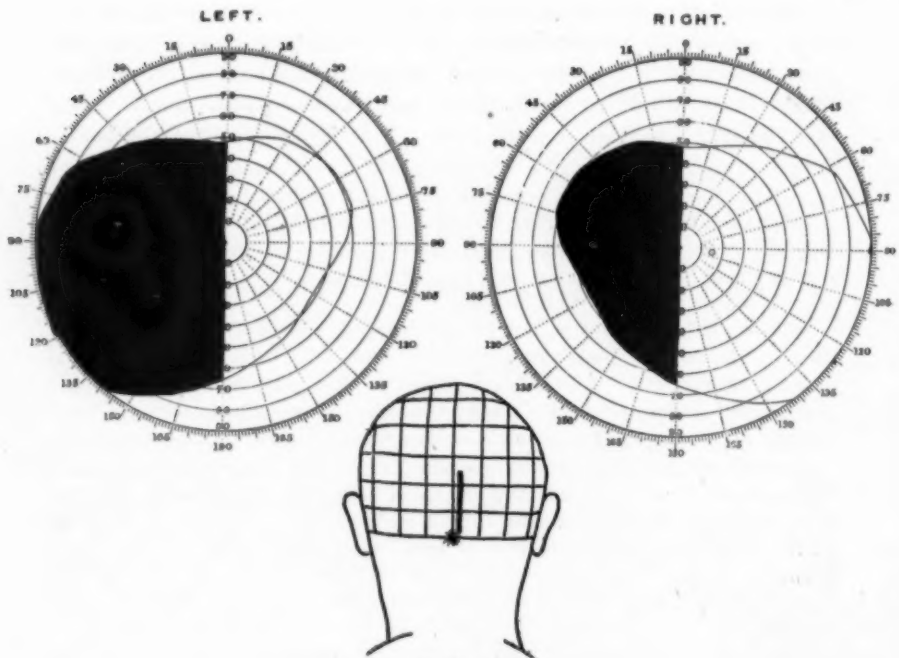


FIG. 22 (Case 19).

be explained by the varying degree of anastomosis between, and the relative extent in the distribution of, the posterior and the middle cerebral arteries at the occipital pole of the hemisphere. The frequent persistence of central vision in bilateral hemianopia due to bilateral cortical lesions may be possibly explained by this same fact.

To make our conclusions complete it would be necessary to bring forward evidence on the cortical representation of peripheral vision. If the macular centre lies in the posterior part of the area striata, and if, as we find, lesions here do not affect peripheral vision, it might be assumed that the latter is represented in the more anterior part of the visual zone; and the fact that the size of the scotoma, whether central or paracentral, varies directly with the depth of the wound from the occipital pole, makes it probable that the concentric zones of the retina from its centre outwards are represented in serial order from behind forwards in the visual area.

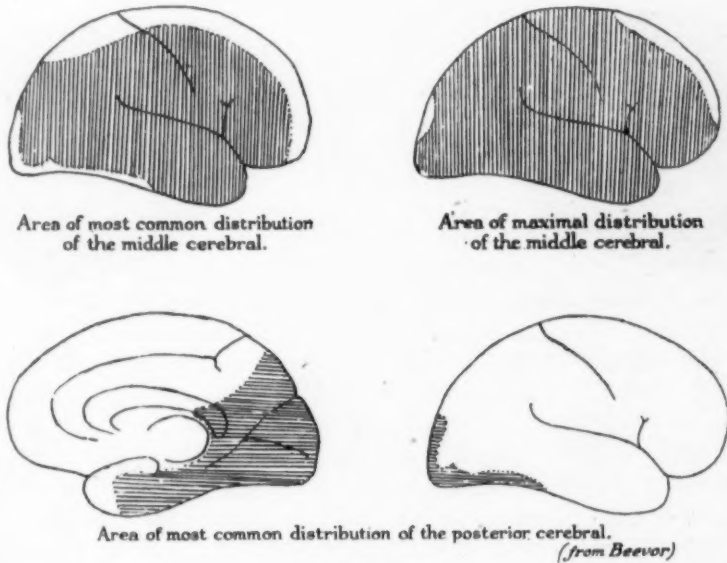


FIG. 23.

We have not had, however, the opportunity of observing any positive case that bears conclusively on the cortical localization of peripheral vision. This is not surprising, as penetrating or perforating wounds which could directly involve the anterior portion of the calcarine region are very liable to injure at the same time the optic radiations. It is not uncommon, however, to find the so-called "telescopic vision" in patients who have been shot through the head in the lower parietal or temporal regions, as in the following case:—

JU—***

Case 20.—Private F. was wounded probably in the middle of June, 1915. He was unconscious for some days and was admitted to a base hospital on June 18.

Wound.—The entrance was 2 in. above and $1\frac{1}{2}$ in. behind the upper margin of the attachment of the right pinna; the exit was $\frac{1}{2}$ in. above and $2\frac{1}{4}$ in. behind the attachment of the left pinna. Both were small, punctured wounds. He was in a very drowsy and critical state when admitted, and had

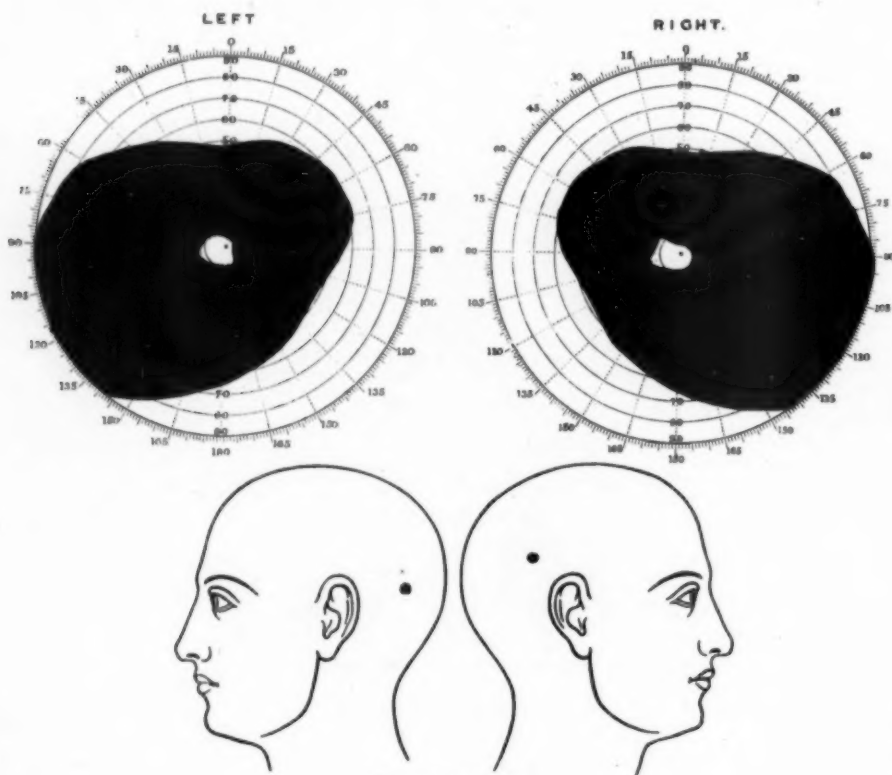


FIG. 24 (Case 20).

papilloedema with swelling of the disc of about 3D. and small hæmorrhages in its neighbourhood, but he improved rapidly after a decompression operation had been performed. The wounds healed rapidly and the papilloedema disappeared, but his vision was then found to be seriously affected. Six weeks after the infliction of the wound he could only read large letters, and

his visual fields were limited to a zone of about 10° around the fixation point (fig. 24). He was able to recognize colours by macular vision. In this case measurements show the bullet probably passed the middle line of the skull, posterior to the splenium and through the anterior part of the area striata, but as the optic radiations must also have been injured, the interpretation of his visual disturbance is doubtful.

It might, perhaps, still be argued that the evidence is not sufficient to conclude that central vision, that is, that subserved by the fovea centralis, has not a double representation in the cortex, as certain of our fields may show that the blindness, whether in the form of a scotoma or of hemianopia, did not extend up to the fixation point; but we have found that the more exact the methods we employed, and the greater the care taken in making the observations, the nearer did the loss approach the fixation point. We consequently believe that, in common with every other part of the retina, the macula is not represented bilaterally.

In a certain number of the visual fields reproduced the areas of blindness are not quite symmetrical in the two eyes; in dealing with central scotomata this may be due to the obvious difficulty in obtaining central fixation, and in other cases to failure or fatigue of attention on the part of the patients suffering with recent head wounds. This tendency for attention to fail generally necessitates frequent short sittings in order to obtain accurate records.

Another point of interest is whether vision for colours may be dissociated from that for white—if, in other words, an achromatopsia may be produced by cerebral lesions. It is true that in certain cases we found the perception of colour lost in areas in which white was visible, but in such regions of the visual field vision even to white was indistinct and uncertain, especially when a small test object was employed. It consequently seems that loss of colour vision for objects of the ordinary test size (10 mm.) may be only a part of general reduction of visual acuity; we can, however, only state at present that we have no conclusive evidence that achromatopsia, with intact vision for white, is produced by cerebral lesions which involve either the cortex or the optic radiations.

Such observations as those we put forward here will be, no doubt, multiplied during the course of the War, and when finally collected should furnish fuller and more definite conclusions on localization within the cortical visual area. Inouye has attempted this from his

observations on men wounded in the Russo-Japanese War, and Dimmer, Uththoff and Axenfeld have already described a certain number of important cases seen during the present conflict.

While this paper was in preparation the valuable and elaborate contribution of MM. Pierre Marie and Chatelin to the same subject has come into our hands. We need only add that our work seems to be mutually confirmatory.

Our conclusions, which cannot yet be regarded as final, may be formulated concisely :—

(1) The upper half of each retina is represented in the dorsal, and the lower in the ventral part of each visual area.

(2) The centre for macular or central vision lies in the posterior extremities of the visual areas, probably on the margins and the lateral surfaces of the occipital poles.

(3) That portion of each upper quadrant of the retina in the immediate neighbourhood of, and including the adjacent part of, the fovea centralis is represented in the upper and posterior part of the visual area in the hemisphere of the same side, and vice versa.

(4) The centre for vision subserved by the periphery of the retinae is probably situated in the anterior end of the visual area, and the serial concentric zones of the retina from the macula to the periphery are probably represented in this order from behind forwards in the visual area.

Finally, it is our pleasure to acknowledge gratefully the help and assistance we have received from the medical officers of the various hospitals in which the cases were observed, and to offer our thanks to Captain Curtis Webb for his aid in the preparation of the illustrations.

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REMARKS BY COLONEL LISTER.

Colonel LISTER: It is only owing to the characteristic generosity of Colonel Holmes that my name is associated with the authorship of the paper. It is true that I have assisted in taking the greater number of the fields of vision, but the main work of looking up the literature and preparation of the paper has been done by Colonel Holmes, and I cannot lay claim to any credit for the various original suggestions which have been put forward.

DISCUSSION.

The CHAIRMAN (Sir Anderson Crichtett, Bt.): We owe a debt of gratitude to our two Colonels for the infinite pains that they have taken to place this very interesting subject so accurately—I might almost say so exhaustively—before us. They have given us so much food for thought that I think our mental digestion may, for some time to come, be rather heavily taxed in dealing with the various hypotheses which they have submitted. Two dominant facts seem to strike one at once: One is the wonderfully increasing power and success of brain surgery, and the other is the marvellous recuperative power of the *vis medicatrix naturæ*. This terrible War has brought but few compensations, but amongst those which have come, I think we may venture to claim the extraordinary opportunities for increasing our knowledge and experience which have been given to the members of the surgical and medical profession; and I think that ophthalmologists are getting their fair share. For many years before the War, the ophthalmic vineyard had been so full of earnest and accurate observers that it gradually became a matter of common comment—I will not say that our subjects were becoming absolutely exhausted—that food for original thought was becoming more limited, and as repetition always involves dilution, there was a little diminished interest. In fact, we were rather drifting in the direction of the small boy,

who, having eaten all his strawberries, had to comfort himself with the reflection that he could still suck the stalks. But now this War has opened up fresh channels for thought and for investigation. One point which has been brought home to us specially to-night is this: that more and more, with regard to our recent experiences, they go hand-in-hand with those of our colleagues the neurologists, and I am glad to know that several distinguished members of the Neurological Section have accepted the invitation which we tendered. I am sure you will echo my desire that they will give us the advantage of their views to-night.

Lieutenant-Colonel R. H. ELLIOT: It is not, Sir, that I do not share the general evident modesty and disinclination to offer any criticism on the paper that I rise, but I desire to take the opportunity of asking two questions which, I think, are of considerable importance. There is a great disinclination—among the younger officers, at all events—to wear the steel helmet, and I would like to ask Colonel Lister and Colonel Holmes whether they have any experience to confirm the estimate which was given officially and in the papers, that the steel helmet has been a great protection to the men and officers in regard to head wounds. It has been said by some that the steel helmet is a death-trap; that it opens up the bullet, and in that way makes the wound more serious. Many of us have boys out there, and some of us are able to influence other young men, and it would be of great interest if we could have opinions from the authorities who have addressed us to-night. The second question I want to ask is this: Combatant officers tell us that there are three periods in the flight of a bullet. They say that up to 250 yards of its journey it has not "settled down," and that within that distance they are humming as they fly over. From that range to 1,000 yards or more, the bullet is said to be travelling quietly and, as they term it, "sweetly"; consequently the wound at such a range is much less severe than it is earlier or later in the flight of the bullet. Most of us who have seen the wounds are astonished at the healing of them, and I have had one or two cases of brain wounds in which the bullet evidently went straight through the brain, but one could find nothing to show for it. The explanation offered has been that it was inflicted by a bullet travelling at its most happy period. Is this view founded on good data? I hope I have not gone off the main track, but if the authors could give us an opinion on the questions I have raised it would, I think, be of great interest to many of us.

Mr. LESLIE PATON: There is, of course, on the part of all of us, a great unwillingness to discuss a subject on which we are profoundly ignorant. I am rising mainly in order to point out something which we on this side can do to help this work. Obviously Colonel Lister and Colonel Holmes see these cases

in the early stages, and they pass away from their care with the subsidence of the shock and perhaps the œdema, and are transferred to civil care in this country; and it is our duty to follow up, as far as possible, the cases which the two authors have seen on the other side. I think those of us who are interested in these cases will receive from what we have heard to-night renewed encouragement to work out fields of vision, for it is only by following up these cases in the later stages that such a thesis can be properly supported. I think we owe it to men who have done such excellent work as has been presented to us this evening to try to do that. There is one matter on which I feel sure Colonel Lister and Colonel Holmes will agree—namely, that if the fields are going to be taken carefully, the mechanical self-registering perimeter must be given up; it is the ruination of field-taking in this country. It is only when taken properly with the perimeter, and especially with the Bjerrum screen, that the fields will be of value for basing conclusions upon. The fields must be taken in this country as carefully as the authors have taken them in France. I have seen several of these occipital injuries, and one or two points of interest have already come out. One is, that in some of the cases in which there has been complete hemianopia, it differs from the clinical hemianopias we have been accustomed to see from the fact that almost invariably the loss cuts straight through the fixation point; there is none of the sparing of the macular field which one finds in so many clinical hemianopias. That is a point on which there has been some discussion. It is often found, in the hemianopias met with in civil life, that if the fields be taken early enough, the macula is not spared, any more than in these military cases. If you get a paralytic hemianopia with hemiplegia, and take the fields early, there may be seen the same clean cut through the middle of the macula; and it is only later on that you find macular vision restored in a small area of two or three degrees. I have seen two or three cases lately, in one of which I took the fields last Sunday. The fields had been taken earlier by Colonel Holmes, and there had been a clean-cut hemianopia. There was a definite recovery of the field, very markedly for white. There was absolutely no recovery of the hemianopic field for colours; the colour field still went straight through the middle of the fixation point. If this discussion results in inducing us to help Colonel Lister and Colonel Holmes to trace out the later history of these cases, and to work out the visual fields after all the shock and inflammatory symptoms and the œdema have subsided, and to discriminate when the visual loss may be expected to represent the purely destructive lesion, it will have done much good.

Mr. J. H. PARSONS: The chief points which occurred to me were those already mentioned by Mr. Paton. I should like to add that Colonel Lister and Colonel Holmes could greatly assist us on this side if they would have the notes of the cases sent over to England with the patients.

Lieutenant-Colonel GORDON HOLMES (in reply): I rise chiefly to thank you for your courtesy in allowing us to place our views before you this evening, and for the very patient way in which you have listened to a paper which, I fear, was too long, and, in places, rather uninteresting. There are, I think, relatively few points to which I need reply. With reference to steel helmets, as Colonel Elliot knows, they have been served out pretty generally to the troops only within the last few weeks, and so there is not much experience to relate in regard to them in our armies as yet. But only a relatively small proportion of the wounds we see at the base are due to rifle bullets; the majority are due to shell fragments, shrapnel, and, above all, to bombs, hand grenades, rifle grenades and aerial torpedoes. There is no doubt that many of the very unfavourable wounds due to shell-casing and to bombs are obviated by steel helmets. We can only bear out what Mr. Leslie Paton has just said, especially with regard to the methods of examination. There can be no doubt that mechanical registering is not satisfactory, especially on the McHardy perimeter. We have where possible used the modification of Bjerrum's screen described in the footnote (p. 58).

Section of Ophthalmology.

President—Mr. PRIESTLEY SMITH, F.R.C.S.

(June 14, 1916.)

Case of Gunshot Wound involving Visual Centre, with Visual Disorientation.

By LEWIS R. YEALLAND, M.D.

(Communicated by LESLIE PATON, F.R.C.S.)

PRIVATE M., aged 31, a maltster by trade, was admitted to the National Hospital, Queen Square, on February 13, 1916, with a history of a perforating bullet wound of the head. Immediately after being put to bed he suffered from a genuine epileptic seizure, but a recurrence has not been observed since. For several hours after the attack he was drowsy and inaccessible.

On examination he was found to have a right hemiplegia with increased reflexes and a plantar response extensor on that side. There were also present various motor and sensory disturbances which have more or less remained, but the hemiplegic condition has since improved considerably, the reflexes now being about normal on the two sides and the right plantar response of the flexor type. Power has returned to the right leg and he can now walk fairly well, but the movement of the right upper limb is still markedly impaired. The left eye appears to stare while the right eye appears normal. There is a left hemianopia. The visual fields are difficult to examine in view of his inability to keep the eye on the fixation point. Mr. Leslie Paton has carefully examined the fields, both of which show marked contraction. All ocular movements are well performed, there being no nystagmus or squint present. The disk edges are quite clear, and there is no evidence of papilloedema. When he is asked to open the eyes the left half of the forehead wrinkles

unduly and in ordinary expressional movements there is flatness of the right side of the face. The expression is stereotyped. The tongue protruded straight and there is no deviation of the palate. Articulation is slow and monotonous, the speech is somewhat nasal. Hearing and taste are unimpaired. It should be mentioned that the patient is right-handed. Examination of the motor system shows weakness on the right side of the body. When he holds out the right hand it falls away and he is apparently unconscious of its movement. There is no limitation of movement, but the general weakness, which is largely due to sensory loss, tends to hinder co-ordinate purposive acts. There is a fine rhythmic, but not regular, tremor frequently developed in the right hand which does not present itself in action. The fingers of the right hand tend to claw or to extend irregularly in the exercise of any action that requires force. The left arm is slightly flexed and adducted. Fine tremors such as are already described in the right arm are present in the left. Tremors develop chiefly when he sits up. In the movements of the left leg it is observed that there are no tremors except on completion of an act. On attempting to maintain an attitude the limb becomes tremulous. Movements of the right leg are inco-ordinate owing to sensory loss. On standing he places the right foot irregularly on the floor and immediately after doing so relieves that side of the weight. When told to get into bed and lie down he falls transversely across the bed and has much difficulty in righting himself to get into bed properly. Sensation: There is sensory loss in the right hand and right leg. In the right hand he misses some contact with cotton-wool. There is also present a relative anæsthesia to pinprick and hot and cold test tubes. Sensory loss of a like nature occurs in the right leg from the knee downwards. There is complete loss of sense of position and appreciation of passive movements in the right arm and leg. There is no appreciation of size and shape in the right hand. Compass test: The left hand gives a perfectly good reading with 1 cm.; the right hand, however, is quite hopeless at 5 cm. to $7\frac{1}{2}$ cm. The sole of the left foot shows no disturbance at $3\frac{1}{2}$ cm., but in the right there is a defect at 10 cm. to $12\frac{1}{2}$ cm. In view of his condition it is impossible to elicit satisfactorily any tests for localization.

The wound of entry is quite obvious, as there is a large pulsating mass situated 6 cm. above and 4 cm. to the right of theinion. The measurements of the wound are 7 cm. in its largest diameter, which is horizontal and 4 cm. in its greatest vertical diameter. No fragments remain in the brain and the edges of the bone are smooth. The exit

is to the left, 16 cm. above and in front of theinion and 5 cm. to the left, about half way between the tip of the left ear and a line drawn from the nasion to theinion. It has the appearance of a clean wound with linear fissures radiating from it about 2 cm. in diameter.

Various tests were performed to demonstrate the visual disorientation which is manifested in this patient. In spite of the fact of his seeing objects he is unable to tell correctly their relation in space, that is when two articles are placed in front of him he is unable to tell correctly which is to the right or left, which the higher, &c., but as a general rule the object to the left of him appears to be nearer to him. Objects remain but a moment in his vision.

Apraxia is also present to some extent, that is he is unable to perform certain purposive acts, in spite of the fact of there being no motor or sensory loss or inco-ordination in the left arm.

Mr. LESLIE PATON: I asked Dr. Yealland to bring this case forward, especially as a very interesting article on a case of visual apraxia was published in the *British Medical Journal* on March 25 this year, reported by Captain Smith and Lieutenant-Colonel Gordon Holmes. In that case there was a similar loss of orientation, and as this type of case has not hitherto been very common, I thought members would be interested in it. To enter into all the details concerning this particular patient would take a long time, though not so long as the elucidation of all the details took. His power of response to any question is far from rapid: even taking the visual fields is an afternoon's work. A very obvious condition which he has is loss of spatial perception. Yet there is no agnosia: he can tell at once the name of any object held up. If one holds up a cup and a saucer, though there may be a difference of half a metre between them in their distance from him, he is quite unable to appreciate it. Only recently, in a series of tests which we tried with him, we found that in the great majority of cases he called that object the nearer which happened to be on his left. He has left hemianopia, that is to say, he is blind on the left side. During this series of tests when he mostly said the nearer objects were on his left, we varied the distances of the objects a good deal. Dr. Yealland tells me he has again tested him in the same way, and again he mostly said the nearer object was on his left. When the objects to be tested were lying on a table in front of him and seeing them required a slight downward movement of the eyes, I thought his answers were more accurate than in the previous tests. Indeed, he seems to be becoming more accurate in his judgments, though he still makes gross errors. In the course of the various tests to which he has been subjected he seems to be re-educating himself in some respects. For instance, he has become more accurate in placing coins on the top of one another. That, however, is partly due to the fact that now when he is asked to do an action he feels the position of the objects: he feels for the penny rather than

tries to see it, so that it is not because his visual orientation is improving, but because, knowing his visual orientation is bad, he tries to compensate for it by his feeling which is good. The movement of his left hand is good, his right hand is hemiplegic. His eyes have perfectly good movement. I thought at first that he had defective movement to the left and that the increased nervous effort to look to the left might be the cause of his false projection, but that is not so. The lesion is, I think, fairly definite. What has taken place is complete destruction of the right occipital cortex: the whole visuo-sensory cortex on the right side has been destroyed. The left visuo-sensory cortex has escaped fairly well. The bullet has passed at the top of the corpus callosum and fairly high up on the left side, just on a level with the top of his ear. The result is, that there has been almost complete severance of the superior longitudinal commissural fibres—those which connect the visuo-sensory tracts with the frontal lobe and front parts of the parietal lobe have been cut through, and there is a loss of that communication on which his power of moving himself in space, and of projecting things into space, seems to depend. So it has evidently been a lesion missing the internal capsule. There may be some fibres of the optic radiations cut through, though there cannot be many damaged as his field of vision is fairly good on the side which is not hemianopic. It is difficult to be sure that the fields have been properly taken, but they seem fairly good. It is difficult to say anything about prognosis. He is certainly improving. If an object is placed in front of him he may see it, but he goes walking straight on. Since coming to this meeting I have heard that Lieutenant-Colonel Holmes has seen a later and exactly similar case.

(June 14, 1916.)

Case of Mikulicz's Disease.

By G. H. POOLEY, F.R.C.S.

(Demonstrated by A. W. ORMOND, Captain R.A.M.C.)

R. A., MALE, aged 58, gardener. First seen on January 26, 1916; sent by Dr. Lind Walker, of Doncaster.

History: He had some eye trouble a year ago, which got well under treatment with a lotion. Eyes well in July last; since then the condition has gradually developed; no pain, he feels drowsy.

Present condition: Marked swelling of both lids of both eyes, with some proptosis of the eyeballs, most marked on the left side; limitation

of movements of eyes in all directions. Vision: Right $\frac{6}{18}$, left less than $\frac{6}{60}$; no improvement with glasses. Right pupil active, left sluggish to light. There is a considerable swelling of the submaxillary gland on each side; the left pre-auricular gland is enlarged. The masses in the eyelids are firm to touch, and mobile; they encroach on the space between the conjunctiva and the left eyeball above. His temperature is 100° F.

February 6, 1916: Operation on left orbit; growth removed as freely as possible. It was necessary to dissect out all the muscles from the growth which filled in all the lymph spaces in the orbit. It was very dense near the apex of the orbit. It was difficult to get away the growth, although it was not adherent to the other tissues. Recovery smooth. Vision of the left eye before operation = fingers.

April 14, 1916: Some ocular movement is returning. Vision: Right $\frac{6}{12}$, left = fingers.

May 9: Right $\frac{6}{18}$, left $\frac{6}{36}$.

May 30: Right $\frac{6}{18}$ partly, left $\frac{6}{18}$.

June 14: Right $\frac{6}{36}$, left with + 1 cylinder $120 = \frac{6}{24}$.

There is some ptosis of his left upper lid, which is improving; ocular movements are limited, but are improving. There was marked improvement in his condition while in hospital; the swelling of the right orbit subsided considerably. He was, and is still, being treated with sodium cacodylate. Since he left the hospital the condition of his right eye has become worse. There is now some return of the new tissue on the left side, principally under the ocular conjunctiva. The operation included turning out the outer wall of the orbit, and the resulting scar is quite free from any invasion.

His blood was counted, and the findings were: Red cells, 5,400,000; white cells, 15,000; hæmoglobin, 10 per cent. A differential count was being made, but the physician who was doing it was sent abroad on Army work.

Professor J. S. C. Douglas, of the Sheffield University, has kindly examined the material, which shows the microscopical appearance of a small round-celled sarcoma.

A blood count made at Guy's Hospital gave the following result: Leucocytes, 13,600: of which in a count of 300 cells there were found to be: polymorphonuclears, 72 per cent.; eosinophiles, 0.3 per cent.; lymphocytes, 19.6 per cent.; hyalines, 7.6 per cent.; basophiles, 0.3 per cent.

DISCUSSION.

Mr. LESLIE PATON: Members may remember that about eighteen months ago I showed a case of this so-called Mikulicz's disease in a woman, very similar to this case. She had the same involvement of the upper lid as in this case, the same protrusion of the eyes, but she had not the same involvement of the lower lids as this man has. She had very marked enlargement of the submaxillary glands, and her palatal glands were also affected, much more markedly so than in this case. I showed specimens of my patient's blood, which might well have passed as typically that of lymphatic leukæmia. The further history of that lady was, that I removed the glands from the upper lid, but I was too late to save the sight of the right eye; members will remember that the cornea of that eye had been very much broken down by exposure. I saved the right eye, but not its sight. The left eye we saved, and the sight of it also. She improved very considerably while under treatment in the Hospital, but, unfortunately, she died four months afterwards of some different disease, when a considerable number of glands which were not palpable during life were found to be affected. The cells infiltrating these glands were very similar in nature to those in the blood. Had I known that this case was to be shown to-night, I should have brought my photographs and blood specimens. The reason I discuss the case is to ask the question whether one is justified in separating this type of disease and calling it by the name of Mikulicz's disease, because the investigation which has been made into the condition has pointed to it belonging to the class of lymphatic leukæmias. By giving it this special name we are apt to be misled in our judgment and as to the appropriate treatment. I do not think it should be regarded as a special entity, and I would suggest to Mr. Pooley that it is desirable to get rid of those affected glands; it is easy to do so. In my case, of which I have spoken, the orbital glands shelled out very easily indeed, and the improvement in her appearance was immediate and marked.

Lieutenant-Colonel ELLIOT: When Mr. Paton showed the case he has referred to, he quoted a case of mine of Mikulicz's disease. I can confirm one of the points made by Mr. Paton—viz., the remarkably easy way in which the glands shelled out. In my case, after removal of the large orbital glands, the other glands in the head and neck very distinctly diminished in size—the submaxillary, the parotid and the sublingual glands. Mr. Paton appears to think that cases of Mikulicz's disease should not be regarded as a clinical entity. I should like to submit a side of the question against that view: (1) all the cases described by Mikulicz and those which have since been described by others have started in one set of glands, in connexion with preceding trouble in a mucous membrane; in my case it started in the conjunctiva: (2) as I have said, the removal of the offending glands causes marked improvement in the remaining glands; and (3) the general health of the patient does not suffer. I do not think that the same can be said to be the rule in lymphadenomatous cases.

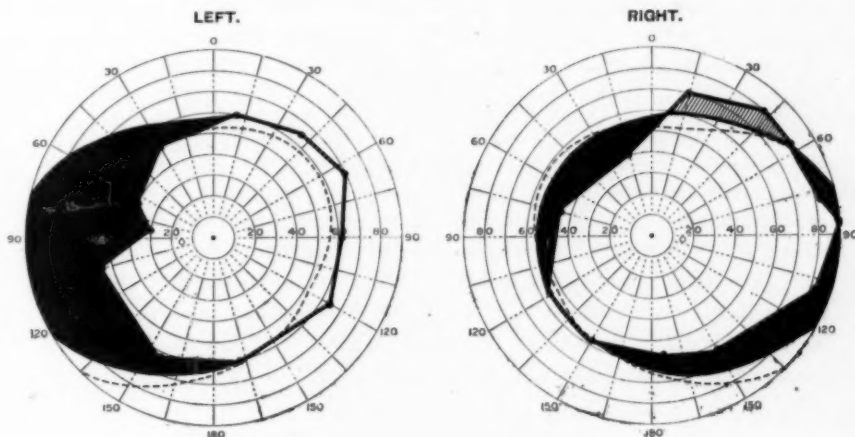
(June 14, 1916.)

Case of Retinitis Pigmentosa (Atypical), with Œdema of the Macula.

By J. F. CARRUTHERS, M.D., Captain R.A.M.C.

PRIVATE J. W., Army Service Corps, aged 19.

Family history: No trace of disease in parents or grandparents; no consanguinity of parents; one sister, aged 24, and one brother, aged 17, suffer from night-blindness.



Vision: Right eye $\frac{6}{12}$, left eye $\frac{6}{12}$. Fields of vision: Both slightly contracted peripherally, the left more so than the right. No albumin, no sugar, no casts. Wassermann test negative. Blood-pressure a little elevated (140). Heart normal. Left fundus shows very little abnormality, but retinal atrophy. Right fundus: Optic disk—Details quite clear, but surface raised about +1.5D. above adjacent retina; macular region œdematous and showing innumerable glistening striæ, between macula and optic nerve especially; periphery atrophic, and in one spot, towards the nasal side of the fundus, three or four faint but definite, pigmented, corpuscular figures are visible in proximity to the vessels.

The lad told me he had been a soldier only three months, and had

not been abroad. Whatever previous conditions have entered into the case, they were only those of an ordinary Lancashire weaver. He has been night-blind since early childhood, and has a younger brother and an elder sister who both suffer from night-blindness. From the patient's description of them, they both seem to suffer from ordinary retinitis pigmentosa; I have not seen them. This lad joined the Army Service Corps, and found, when he was put to do driving, that he was quite capable during the day, but, on the approach of dusk, he became useless, and reported himself sick, and was sent to me. The first thing which struck me on looking into his eyes was a gleaming condition about the macula of the right eye. After dilating the pupil I thought it was an oedematous condition of the macula. Mr. Paton, who looked at it, also thought there was a little congestion of the right papilla. The periphery showed hardly any changes of a noticeable kind. There was slight retinal atrophy, until we discovered two or three small pigmented corpuscles of the usual retinitis pigmentosa kind, on the nasal side, towards the periphery of the right eye. At the beginning of last week these corpuscles were three in number, but to-night there are four or five. Otherwise there is no definite sign of retinitis pigmentosa in the case, and, as most of you know, it is unusual to see oedema of the macula or any acute process in retinitis pigmentosa; I have never seen that condition in a case of retinitis pigmentosa. It is usually a quiet, practically atrophic process. The interest of this case lies in the fact that there seem to be acute symptoms, and the oedema of the macula I regard as unusual. I shall be glad to hear what the members who have seen it to-night think of it.

DISCUSSION.

Mr. LANG : I notice fine floating opacities in the vitreous which I consider is additional evidence of its being a case of retinitis pigmentosa.

Mr. STEPHEN MAYOU : I examined this case very carefully, but I have never seen macular changes such as are present in this case, in retinitis pigmentosa. If this is a case of retinitis pigmentosa, it is at such an early stage that one would not expect to find changes in the macula. I think the fields hardly bear out the diagnosis. There is peripheral contraction, but there is no ring scotoma present. Nor do I think the pigmentation is quite typical. Therefore I regard the diagnosis as somewhat doubtful.

Mr. J. B. LAWFORD : The case is puzzling, in more ways than one. The boy's personal history and the family history are both in favour of the diagnosis of retinitis pigmentosa, and the boy says he has *always* been night-blind.

That, combined with a similar history in two other members of the family, is strong presumptive evidence in favour of retinitis pigmentosa, or of some familial condition of which night-blindness is a prominent symptom, not necessarily retinitis pigmentosa. The appearances in the right eye are unlike those of ordinary cases of retinitis pigmentosa, they rather give one the idea of something more or less acute. In the left eye the conditions are again different, and my impression is that if it be a case of retinitis pigmentosa there is some acute condition superimposed.

Mr. LESLIE PATON: I suggested to Captain Carruthers the diagnosis retinitis pigmentosa in this case, with changes at the macula. I thought that to be the simplest way of classifying the case for the time being, considering both the family and personal history. I still consider that is what it is. But if it had been an ordinary case of retinitis pigmentosa, I should not have advised Captain Carruthers to bring it up for exhibition. The pigmentation here, though slight, is definite and is becoming more marked; it is taking on the typical corpuscular form, and it is appearing in the equatorial region. I did not see the fields till to-night, and I was surprised to find that they were not of the ring scotoma type. But what made me hunt more definitely for pigmentation—I did not see pigmentation the first time I looked for it—was the fact that there was obvious retinal atrophy: the peripheral portion of the retina has that peculiar brown dry-leaf appearance which one sees in retinitis pigmentosa *sine pigmento*. From the evidence we have, this peripheral retinal atrophy must have been of considerable standing, because the boy has been night-blind for a considerable time. I do not know why there should be superimposed on that retinal atrophy this acuter condition, which is shown by the presence of the oedema at the macula. The oedema is variable, because it is not so marked now as it was a week ago. Then one could see radiating folds, and oedema reaching from the macula to the disk edges. I agree with Mr. Lawford that in this case there is probably some acute process overlying the atrophic process. Whether the two are associated, and if so, what the nature of that association may be, I cannot say.

(June 14, 1916.)

Light Spot Perimeter.

By A. C. HUDSON, M.D., Captain R.A.M.C.

THE test object consists of a white or coloured square of variable area, which is made to travel over the concave surface of a broad quadrant arc of one metre radius, either by direct action of the hand acting from behind the arc, or by rotation of a wheel at the axis of rotation of the arc. With any movement of the test object a corresponding

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movement on a reduced scale is communicated to an electric lamp travelling on the posterior surface of a plane extension of the arc distal to its axis of rotation. A beam of light from this lamp passing through a pin-point diaphragm situated immediately behind the axis of rotation produces a small area of relatively bright illumination on a paper chart which, stretched over the posterior surface of a sheet of glass at a short distance behind the diaphragm, is elsewhere thrown into relative shade by the portion of the diaphragm surrounding the aperture. A reading is recorded on the chart by means of a pencil mark or pin-prick placed at the centre of the light spot. By means of a change of gear, so arranged as to communicate to the lamp a movement of similar extent to that of the test object, abnormalities in the central part of the field of vision, including the region of the blind spot, may be charted on a correspondingly enlarged scale. The instrument is made by Messrs. Dixey.

I apologize for showing the apparatus before it is complete in all its details, but I was anxious not to miss the opportunity of obtaining criticisms and suggestions as to improvements in construction afforded by this last meeting of the Session. The completed instrument will be provided with an alternative gear, so that the central region of the field of vision, including the blind spot, may be mapped out on a larger scale than the peripheral field.

Lieutenant-Colonel ELLIOT: I have been actually working with a perimeter on the same lines for six months. The difficulty I had originally, which I do not think Captain Hudson has overcome, is that of getting a strong enough arc. At first I had an arc something like his, but I found it altered its curvature, so I had to make one of a stronger piece of metal in order to retain the correct curve. That, of course, made the apparatus heavier, and I think Captain Hudson will have to use a more stable base. I had to drive plugs into the wall before it was stable enough. With regard to the registration arrangement, you, Sir, have criticized it on the grounds (1) that first of all one gets a large record of a scotoma and then afterwards has to reduce it on the chart; and (2) that you doubted if it was easier to chart a scotoma with a large perimeter than with a small one. I have several perimeters, and I find that working with a large perimeter is vastly easier for the patient—whatever it may be for the surgeon—because the patient can discern a very small scotoma, or map out the blind spot, with an ease which is not attainable when working at closer ranges. Your objection, that the advantage gained is thrown away, to a large extent, by using a small chart I found was a valid one. In order to meet it, I have had specially large charts made, 8 in. in diameter, and I dictate, as I go along, what the readings are, whilst a nurse takes them down and transfers them to the chart afterwards.

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The Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.

Otological Section.

President—Dr. ALBERT A. GRAY.

(November 19, 1915.)

**Bony Sequestrum, believed to be the Annulus Tympanicus,
removed from the External Meatus of an Infant, aged
2 Years 9 Months.**

By H. L. WHALE, F.R.C.S.

THE child has suffered continuously from otorrhœa since the age of 14 months.

The specimen exhibited broke this afternoon, so members cannot now see what it looked like. I do not know whether it is an annulus; I showed it to Dr. Dundas Grant, and he agreed that was what it appeared to be.

(November 19, 1915.)

**Otomycosis; Colony and Microscopic Slide of *Aspergillus
fumigatus*.**

By H. L. WHALE, F.R.C.S.

THE fungus was growing profusely on cerumen on the floor of a radical mastoid cavity in a male, aged 25. The cavity was entirely epithelialized and dry, not eczematous. The patient complained of slight itching. The mass looked like frost-covered grass, white, with an obvious glint of green in patches. The patient had not been living

in a hot climate or unhealthy surroundings, nor had he been putting oil into his ear, nor been in contact with pigeons or other birds.

The specimen was kindly prepared by Dr. St. John Brooks.

DISCUSSION.

The PRESIDENT: Otomycosis sometimes presents a very curious colour. I saw one case of a brilliant emerald green. There was evidently some change in the colour of the fungus, probably due to the absorption and transformation of some pigment from the wax.

(In reply to a question asked by Mr. West): I should call this otomycosis. I do not know that the term has been exactly defined; perhaps members will give their definitions.

Dr. GRANT: In old days, when oil was much used in treating ear conditions, otomycosis was said to be much more frequent than now. At that time probably the *aspergillus* grew in the oil rather than in the tissues. In the present case the cerumen may act as the base in the same way. I do not think the fact of the growth taking place on cerumen is an objection to the application of the term "otomycosis."

Professor URBAN PRITCHARD: I have always been much interested in the subject of *aspergillus*. I do not believe the application of oil to the ear had any part in the causation. I was on the look-out for the condition—some twenty-five years ago—for six years before I saw a case, so that it would not seem to have been very common then. It is less common in England than anywhere else in the world. It is common in Switzerland and Austria, and it is the commonest ear condition noticed by doctors in Bombay. I think its incidence is more to do with locality. I have half a dozen specimens of the *niger* variety from my own patients, but fewer of the *fumigatus*. I think the only differentiation between *fumigatus* and *flavus* is derived by growing a culture. I had a specimen of *flavus* sent me from New York, and that was yellow under the microscope. Mr. Whale's culture specimen shows the *fumigatus* colour beautifully.

Mr. WHALE (in reply): In answer to Mr. West's question, I might call it a case of fungus growing in the ear; it did not grow on the surface epithelium itself. This specimen is *Aspergillus fumigatus*, whereas the ordinary one is *Aspergillus niger*, which is met with chiefly in India, where I was for a time. The *fumigatus* is supposed to be specially met with in people who are brought into contact with birds a good deal, especially pigeons; that was stated by the botanist who helped the pathologist to prepare this specimen. I showed the case because of it being the rarer form. This was certainly acquired in England. The only three varieties I could find mentioned in the books are: *niger*, *fumigatus*, and *malignus*.

(November 19, 1915.)

Keratosiis of the Auditory Meatus.

By W. STUART-LOW, F.R.C.S.

A GIRL, aged 8, who has had a discharge from the left ear for over a year. Two months ago white spots began to appear at the lowest and outermost part of the meatus: these gradually coalesced and spread round the passage, forming a tough white ring. Dr. Wyatt Wingrave has examined specimens under the microscope, and describes it as an irritative keratosis similar to what is not uncommonly met with on the tonsils, and said to be due to the presence of a leptothrix. He thinks any true parasitic growth can be completely excluded in this case.

Suggestions as regards treatment are requested.

REMARKS BY DR. WYATT WINGRAVE.

A Portion of Thickened Epidermis from the External Auditory Meatus.—The tissue consists of closely applied hypertrophied papillæ whose superficial squames show two changes—vacuolation and keratinoid granular deposit. The dermal papillæ do not appear to have undergone any change. Between the laminæ of the squames many mycelial threads are seen, but no spores. It may therefore be a mycosis associated with keratosis. Mycelial filaments may be derived from dirty water used as an injection or may be air-borne. Still it must not be forgotten that they closely resemble the micro-organisms of a ringworm. Mycelium alone is, however, strongly in favour of a saprophytic nature. There is no evidence of acute inflammation.

DISCUSSION.

MR. CLAYTON FOX: I think this is a papillomatous condition and that the mycelium is only associated with it, not causally. Such a papillomatous condition is rare. It bears no resemblance to keratosis obturans. It is probably the result of irritation from long-standing suppuration of the ear. I suggest the use of salicylic acid in paroleine, which will act as a solvent for the epidermis and also protect the part from the irritating discharge.

DR. DUNDAS GRANT: This is an extremely rare kind of case, and seems to be a diffuse warty condition. It differs from the classical keratosis obturans

which Wreden described many years ago. I shall be glad to hear whether members have seen anything like it before. I think repeated application of a spirituous solution of salicylic acid would do good.

The PRESIDENT: Sometimes one sees benefit from mechanical measures, such as gauze packing; salicylic ointment can be applied in the gauze packing.

Mr. STUART-LOW (in reply): I thank you for the remarks about treatment, as I was at a loss what to do for the case, though I was inclined to try salicylic acid, if I knew the best strength. When I removed a piece for examination it bled profusely, which would perhaps be an indication against scraping as some members have suggested.

(November 19, 1915.)

Case of Fractured Skull with Healed Attic Suppuration of an Unusual Type.

By E. D. DAVIS, F.R.C.S.

THE patient, an engineer, aged 42, complained of deafness after a crushing injury to the head, in which the vertex of the skull was fractured, and an operation for depressed fracture was performed by Mr. Rowntree on April 24. At the time of the accident there was hæmorrhage from both ears. The left ear shows extensive and healed attic suppuration, revealing a cavity of considerable size above and behind the tympanum. Both external auditory meatus are unusually large in the region of the drum. The deafness is of the middle-ear type, and there is no history of the deafness or of ear disease previous to the accident.

Mr. O'MALLEY: I have not seen the case, but it seems from the description to correspond closely with one which I recently had under my care at Woolwich. The patient was in the motor cycling corps and started out on duty one night and had an accident. He recollected nothing of it until he recovered consciousness forty-eight hours afterwards. On arriving at Woolwich he was complaining of deafness; he also had eye troubles. His fourth, sixth and seventh nerves were damaged, and on examining his ear I noticed a large fissure in the upper part of the tympanic membrane extending into the roof of the tympanum. X-ray examination suggested that there was a fracture in this region.

(November 19, 1915.)

Total Occlusion of the Right External Auditory Meatus.

By L. A. LAWRENCE, F.R.C.S.

THIS condition was noticed in a woman, aged 31, who was sent to hospital on account of pain attributed to the right ear, but in all probability due to a carious tooth in the right lower jaw. The right meatus is occluded by a smooth partition stretching completely across the meatus about a third of the depth from the surface. Careful examination failed to show any opening whatever. There was no moisture nor discharge on the surface. No other deformity was found. The Eustachian tubes are both patent. Râles were heard through a passed Eustachian catheter on both sides. The left ear is normal. The patient hears an acoumeter 2 in. from the right ear. Weber's test indeterminate. Rinne's test: Right ear —, left ear +. Schwabach neutral. Tuning-forks: R. $\frac{9}{8}$, L. 16. Monochord: R. $\frac{17}{12}$, L. $\frac{17}{15}$, with noise apparatus. The condition is evidently congenital, and the case is shown on account of the rarity of the condition.

DISCUSSION.

Dr. KELSON: In 1904 I showed a case before the Otolological Society very much like this in appearance. There the membrane was cicatricial, following suppuration. It is somewhat difficult to exclude that in this case. There may have been some trouble in childhood which has been forgotten. I would like to know what is the differential diagnosis between a congenital condition and one following suppuration. Possibly this may be post-suppurative.

Dr. DUNDAS GRANT: In this case there is absence of a history of suppuration. I had a case not long ago which was almost identical with it, and it was one in which pure carbolic acid had been put in by mistake for carbolized glycerine, and the resulting erosion led to occlusion. The hearing was, however, so much improved by the use of the Eustachian catheter that there seemed to be no justification for cutting through the diaphragm as one would otherwise have been inclined to do, and I think the middle ear behind it was sound. As a rule in congenital cases there is some concomitant defect of development in the auricle or face, or the Eustachian tube. In the present case everything except the diaphragm seems perfect, and I think there must

have been some traumatic cause for the condition early in childhood. My idea of treatment would be to make a small opening in the thinnest part of it and insert a fine tangled tent, small at first and then gradually increased. I think that is more likely to cause dilatation than cutting it into quadrants and stitching up the small triangular portions as is so beautifully described in some of the old text-books. May I ask for an explanation of the numerical nomenclature used in the notes of the case in regard to the monochord?

Dr. DAN MCKENZIE: Last year I showed a case very closely resembling this. There was a definite history of suppuration in the ear during childhood. Probably the present case is not congenital but is produced as the result of ulceration of the external meatus or by traumatic means. The congenital conditions with which we are familiar do not resemble the appearance here.

Dr. H. J. BANKS DAVIS: I thought it was so hard that it might be solid bone. I think a skiagram should be taken of it to see whether there is any meatus behind it.

Mr. CLAYTON FOX: There seems to be a ring of solid bone round the periphery, 1 or 2 mm. in width. I think it is likely to be a congenital case because it is so definite and symmetrical.

Mr. SYDNEY SCOTT (replying for Mr. Lawrence): I think the atresia may be due to adhesions in early post-natal life. The obstructing tissue is not of bony hardness—it can be easily indented with the probe. The tuning-fork tests are carried out with Bezold's fork and the steel monochord. The former figures represent the lowest audible vibration frequency at maximum amplitude; the latter figures represent centimetres and indicate the length of wire used to produce the highest tone audible by air and by bone conduction, the lower of the two figures refers to bone conduction. I think Mr. Lawrence will agree with Dr. Kelson's suppositions.

(November 19, 1915.)

Case of Cartilaginous Overgrowth after the Radical Mastoid Operation.

By W. M. MOLLISON, M.C.

H. C., AGED 15, has suffered from chronic otorrhœa for years, and has had several operations. Two years ago a radical mastoid operation was performed on the left ear. A few months later a cartilaginous

nodule appeared at the concho-meatal junction, and has grown to its present size slowly. Suppuration has continued in spite of operation, and now the mass of cartilage is obstructing the meatus.¹

(November 19, 1915.)

Case of Audible Tinnitus.

By W. M. MOLLISON, M.C.

PATIENT, a boy, aged 9, complained a few days ago of a "ticking" in his ears; his mother noted that she could hear the noise herself. There is an easily audible ticking in the left ear, very clearly heard through an otoscope; it is not synchronous with the pulse, and is much less audible when the boy's mouth is open; a similar noise is to be heard in the right ear, but much less loud. The boy has retracted membranes, and large tonsils, and adenoids. The noise is presumably due to irregular contractions of the tensor tympani.¹

(November 19, 1915.)

Transparencies of Microscopical Sections through the Temporal Bone, from a Case of Otosclerosis.

By A. A. GRAY, M.D.

Four photographic negatives illustrating bony changes in otosclerosis.

¹ Discussion on these cases was postponed to the next meeting of the Section.

(November 19, 1915.)

DISCUSSION ON SKIN-GRAFTING IN MASTOID OPERATIONS.

Opened by H. J. MARRIAGE, F.R.C.S.

MR. PRESIDENT AND GENTLEMEN,—

In the first place, I wish to express my thanks to our Council for the honour they have done me in asking me to open this discussion, and I much regret that, owing to the large amount of time which we are all spending in the treatment of the wounded, I have been unable to refer to the literature of the subject as much as I could wish. I therefore propose to deal chiefly with my own personal experiences, in the hope that other members will speak of their methods and results, so that we can form some definite opinion as to whether skin-grafting is the best form of treatment in these difficult mastoid operations.

In the first place, as regards cases of acute mastoiditis, where the antrum and mastoid cells are opened up without any interference with the tympanum, I think it is quite evident that skin-grafting is not advisable, as the object aimed at is to get free drainage and to allow the cavity formed to fill up as much as possible with granulation tissue and new bone.

The cases for which skin-grafting is, in my opinion, most advantageous are those of chronic mastoid disease, and I propose to devote most of the time allotted to me in discussing the treatment of these cases by skin-grafting, both primary and secondary. As far as I can trace, skin-grafting in the radical mastoid operation was first brought forward by Siebenmann in an article in the *Berliner klinische Wochenschrift* in 1893,¹ who suggested applying a graft some two or three weeks after the primary operation. In 1897 Denker advised in certain cases the application of Thiersch grafts two to four weeks after the primary operation, and in 1903 I saw Jansen applying small grafts with intervals between each piece of skin at the time of the original operation, and I believe he had been doing this for a year or more. I think, however, that the chief credit for this form of treatment belongs to Mr. Charles

¹ *Berl. klin. Wochenschr.*, 1893, xxx, pp. 44, 45.

Ballance, who brought the subject prominently before our profession in the very instructive paper which he read before the Royal Medical and Chirurgical Society on January 23, 1900.¹ In this paper he recommended the application of one or more large Thiersch grafts to the cavity about a week or ten days after the performance of the radical operation. Since that time, many suggestions have been made as to how and when the graft should be applied, and I will now shortly mention some of the different methods.

Ballance's original method was to cut a large Thiersch graft 3 in. by 2 in. from the inner part of the thigh, and, by means of a large section lifter similar to that used in the making of microscopic slides, to transfer this to the mastoid cavity; by the use of a glass pipette the graft was then sucked into position so as to line accurately the whole cavity, a second smaller graft was applied to the soft parts behind the ear, and one end brought through the meatus, this graft being made to cover the raw surface of the posterior meatal wall from which the concho-meatal flap had been cut. Gold leaf, or some other form of protective, was then applied to the surface of the graft, and small sponges with a strip of gauze next inserted to keep the graft in position. As a modification of this method, Milligan proposed "to fill the mastoid cavity with normal salt solution, floating the graft upon the fluid and then sucking up the fluid *per meatum* with a large pipette. As the fluid is withdrawn the graft sinks into the cavity, and ultimately comes to lie in accurate apposition with its granulating walls."² Another method is to use, instead of a lifter, a piece of crêpe tissue, upon which the graft, with its cut surface facing upwards, is carefully spread. The crêpe is then gathered up and, with the graft still clinging to it, is inserted into the operation cavity by way of the auditory meatus and, with the aid of a probe, spread over its walls.

In 1903 I saw Politzer employ the following method so as to avoid reopening the post-aural wound. He made use of a glass tube with a bulb at the end of it, in which were several small holes; a small graft, which was cut under local anæsthesia, was placed over the end of the bulb with the epidermal surface against the glass, and after the bulb had been introduced into the ear through the meatus the graft was blown into position. Several small grafts were employed, and the cavity then plugged. A similar method has also been advocated by Dr. Stoddart Barr.

¹ *Med.-Chir. Trans.*, 1900, lxxxiii, pp. 125-165 [Discussion, p. 173].

² "Diseases of the Ear," by Milligan and Wingrave, p. 398.

I now wish to say a few words with regard to the cutting of the skin-grafts. This, I believe, has caused some surgeons a good deal of trouble, and is probably one of the reasons why, at times, good results have not been obtained, as the grafts cut have been much too thick. The method I employ is to place the thigh in the abducted and everted position, and to put a small sandbag under the lower end of it so as to get a flat surface. My assistant then places the ulnar surface of his hand about 3 in. above the knee and draws the skin downwards towards the knee as much as possible. With my left hand I make counter traction towards the hip, so as to get the skin fully stretched, and then, with a hollow-ground razor which is about half as large again as the ordinary shaving razor, I cut a thin graft about 3 in. long and 2 in. wide, always cutting towards the knee. Various methods have been suggested with the idea of making this part of the operation easier; for example, Waggett recommended painting the skin with "newskin"; Wyatt Wingrave with a solution of celloidin in acetone (10 per cent.), and Deanesly advised applying sticking-plaster to the thigh before cutting the graft, and I believe from America came the suggestion that a piece of board should be used instead of the assistant's hand to keep the skin on the stretch so as to get a more level surface, but I think none of these aids are really necessary after a very little practice.

Having mentioned the methods of cutting and applying the graft, I now wish to discuss the advantages and disadvantages of skin-grafting. The advantages, in my opinion, are:—

- (1) The cavity heals much more quickly than by other methods.
- (2) Contraction of the cavity and stenosis are prevented, and at the same time there is no possibility of granulations extending across various parts of the cavity, and so shutting off cavities which remain unhealed and cause persistent discharge.
- (3) The patient is spared a large amount of pain which formerly was caused by firmly plugging the raw surface of the mastoid cavity.
- (4) Both the patient and surgeon are saved much time and trouble, as about ten days after the grafting operation the patient is able to do nearly all that is necessary for himself and only see his surgeon once a week; and the patient is thus able to return to work much earlier than when he was obliged to attend daily for treatment.
- (5) The surgeon is not worried by having to make complicated meatal flaps, many of which cause much deformity of the concha, and when made are often difficult to keep in position, as all that is necessary is to remove sufficient of the posterior meatal wall to ensure easy access to the antrum and mastoid cavity.

The method I usually employ is to make an incision at the junction of the posterior meatal wall and the concha, and a second incision along the middle of the floor of the meatus, and, after cutting away some of the subcutaneous tissues, to suture with catgut the flap so formed to the soft parts immediately behind the ear, so that the skin surface of the flap looks toward the mastoid cavity.

(6) In a large proportion of cases the Eustachian tube is closed, and reinfection via the tube is thus prevented.

As regards the objections to skin-grafting, it has been pointed out that it is impossible to get the cavity aseptic, but in practice it has been found that this makes no difference and that in 99 out of every 100 cases the graft takes perfectly well, and that even in the exceptional cases, when the grafting is not a complete success, islets of cells are left behind from which the skin quickly grows over the cavity. At the same time I should like to point out that in performing the radical mastoid operation it is essential thoroughly to open up all depressions in the bone, so as to make sure that septic cavities are not left behind the graft; it is also necessary that the graft should be extremely thin.

With regard to the hearing, Ballance in his original paper stated that he found the result much the same as after the older method of dry gauze tamponning, and in a later note added that the result was better than in the older method, attributing this to the very thin layer of tissue which formed over the fenestra ovalis. In a paper published in the *Lancet* on August 17, 1912, Ballance also states that 75 per cent. of a number of private cases tested showed remarkably good hearing as the result of the operation. Grafting certainly has one small disadvantage, and that is that the cavity is very liable to become filled with a collection of cerumen and epithelium which, if left, may cause ulceration of the skin surface; but the collection is easily removed, and, if done regularly once or twice a year, no harm results.

I next wish to consider the question of primary skin-grafting—i.e., the application of a graft at the time of the original mastoid operation. I first tried this method in March, 1908, and since then have used it regularly in uncomplicated cases of chronic mastoid disease; but I do not use it in cases of acute mastoid disease, where it is necessary to perform a radical mastoid operation, preferring in these cases to do the skin-grafting at a later date.

The advantages of the primary skin-grafting are, in my opinion:—

- (1) Seven to ten days are saved in the convalescence of the patient.
- (2) It does away with the necessity of a second anæsthetic, which

many patients so much dread, and I think that having two anæsthetics so close together did affect the patient's general health, especially in the case of those who were very nervous.

The method I employ is as follows : I first perform the ordinary radical mastoid operation, taking care to remove as far as possible every trace of disease, and exposing the dura of the middle fossa and the lateral sinus if I am not quite satisfied with the appearance of the bone lying in contact with these. After cutting a meatal flap I then scrape out the tympanum, removing the whole of the mucous membrane except a very small margin around the stapes, after which the opening of the Eustachian tube is curetted with a small sharp spoon. In order to disinfect the cavity and check the bleeding, I next pour in hydrogen peroxide (20 vols.) which is left in for two or three minutes, and then syringed out with normal saline at 105° F. This is done three times. The cavity is immediately plugged with gauze, the plug being left in while the graft is cut and only removed when everything is quite ready for applying the graft, which is got into position by means of the suction apparatus recommended by Mr. Ballance, and kept in place by the immediate insertion of a long strip of ribbon gauze, $\frac{1}{2}$ in. in width, on which has been dusted some aristol powder to prevent the discharge from becoming offensive. No protective of any kind is used. The end of the gauze is passed through the meatus, and the free end of the graft which is lining the posterior part of the cavity is folded over the plug and also brought out through the meatus, so as to cover its cut edge. After ligaturing the vessels, which have been caught with artery forceps during the operation, the skin opening is sutured, bringing the ear back into its normal position. The plug is left untouched until the fourth day after the operation, when it is withdrawn and the cavity syringed out with a weak solution of hydrogen peroxide, a small piece of ribbon gauze being again lightly inserted to soak up the discharge. The same treatment is continued daily for about one week, when all plugging is stopped, and plain hydrogen peroxide (10 vols.) is dropped in twice daily, and the patient seen at intervals of about seven days, so that any small areas which are granulating too freely can be cauterized with silver nitrate or scraped with a sharp spoon. The superficial part of the graft gradually separates, and usually comes away when the ear is syringed, but it may be necessary to remove it with forceps. When the cavity is nearly healed I often prescribe rectified spirit, or equal parts of hydrogen peroxide (20 vols.) and rectified spirit, so as to harden up the skin surface.

In my opinion, primary skin-grafting is to be preferred to blood-clot dressing, as in the latter method, in addition to the risk of the clot becoming infected and breaking down, the granulations formed in some individuals are liable to become too exuberant, and instead of being transformed into a glistening membrane have a tendency to fill the whole cavity with fibrous tissue, causing obstruction of the round and oval windows. In any case I think it is better to have the cavity lined with healthy skin rather than with cicatricial tissue. With regard to scarlet red and similar substances, the chief objections are that constant attention is required and that pockets are liable to form owing to granulations from different parts of the cavity fusing together. This method may perhaps be useful for the healing of granulating areas when the graft has not taken well, but I shall be pleased to hear the opinions of members who have had a large experience of these methods.

When first performing primary grafting in 1908, I kept notes of the first fifty cases, and I have referred to these to see how long each case took to heal—i.e., for the cavity to become completely covered with skin and absolutely dry. In these cases the plug put in at the time of the operation was left in for seven days instead of four as now, and though I cannot give exact figures for a series of my later cases, I feel certain that my results now are considerably better than they were in 1908, as I pay more attention to the posterior meatal wall, removing as far as possible any exposed portion of cartilage, because I found that this was responsible for the delay in healing in the majority of cases. Of these fifty cases, seven were lost sight of after leaving hospital, as five returned to the country and were not seen again, and two failed to attend for treatment, so I propose to deal with the remaining forty-three. Of these, one healed in nineteen days, one in twenty-one days, six in one month, one in five weeks, seven in six weeks, seven in seven weeks, five in two months, six in two and a half months, one in three months, one in three and a half months, three in four months, one in four and a half months, two in five months, and one in nine months, so that you will observe that thirty-four out of the forty-three cases were healed in two and a half months or under. Of the other nine cases, caries of the internal wall of the tympanum caused the delay in four—viz., one of three and a half months, one of four months, one of four and a half months, and one of nine months; caries of the floor of the aditus in one of four months, and of the posterior cartilaginous meatus in the other four cases—viz., one of three months, one of four months, and two of five months, and in one of these (the case which took four months) the

delay was partly caused by the patient stopping away for two months soon after leaving hospital.

I should like to mention that in these fifty cases, the dura of the middle fossa was exposed in twenty-five, the lateral sinus in two, and both the dura of the middle fossa and the lateral sinus in fifteen, so that in forty-two out of the fifty cases there was exposure of some part of the dura. In no case did I have to remove the graft on account of suppuration.

I have also looked into the important question of hearing, though unfortunately only a watch was employed. Of the forty-three cases which I was able to follow until they were completely healed, I found that two had internal ear deafness before operation, and in two more the original hearing was not noted; I can therefore only deal with thirty-nine. Of these the hearing improved in twenty-nine (74.3 per cent.), deteriorated in eight (20.5 per cent.), and remained unaltered in two (5.2 per cent.).

The results given by Grunert, after the tamponning method were: improved, 55 per cent.; unaltered, 39 per cent.; diminished, 6 per cent.; and by Stacke, who employed the same method: improved, 36 per cent.; unaltered, 57 per cent.; diminished, 7 per cent.

I must lastly say a few words with regard to the cases of chronic mastoid disease where complications are present. If there is a fistula in the bony wall of the semicircular canal, without damage to the membranous canal, I apply a primary graft, and in the series of cases I have just mentioned there were two cases of bony fistula; one was perfectly healed in six weeks, and the other returned to the country at the end of three weeks, when the cavity was healed except for the posterior cartilaginous meatus, which was still granulating. Both cases were relieved from the giddiness with which they suffered before operation.

In cases of suppuration of the internal ear, I do not apply a graft, as the object of the operation is to obtain free drainage, and I think that the application of a graft is very liable to defeat this object.

The PRESIDENT.

We are very much obliged to Mr. Marriage for the clear way in which he has put this matter before us. Historically, I was interested to hear him mention Siebenmann as being the first to employ these grafts, because I saw those cases when I was studying under Bezold at

Munich. With regard to Mr. Marriage's remarks concerning secondary grafting, I tried that method a few times, but never used it much. I gave it up long before primary grafting was introduced, as I was disappointed. Moreover, I found that patients disliked a second operation, requiring a second anæsthetic. Of primary grafting I have not had experience, but I have used a method of my own, which, however, has not yet been published. The method is as follows: Immediately after the mastoid operation, I scrape a considerable area of skin on the forearm, after having made the area aseptic (but not with iodine), and I spread the pulpy mass, consisting of epithelial cells and blood, on to little sponges, or, preferably, on to gutta-percha tissue, as one would spread butter on to bread. They are then pressed down on to the bony surface, the sponges or gutta-percha tissue being removed at the first dressing. At the end of the first week or fortnight one would think there was no difference in the wound compared with an ungrafted one, but after that period epidermis rapidly begins to appear in the form of islands which coalesce, and the whole bony surface is soon covered. With regard to exposure of the dura mater, I always do that purposely, because I expect it to be a more likely site for the grafted material to live upon. With regard to removing the packing on the fourth day after operation, that was the day which I also hit upon quite independently. At first I only allowed two days, and gradually worked up to the fourth, whilst Mr. Marriage worked the other way from a larger number of days. I find the use of scarlet red helps considerably, and it is probably more useful in my method than it is likely to be in Mr. Marriage's; in mine it helps the cells to radiate from the little islets which are formed. I first used scarlet red as an ointment; now I employ it as a powder mixed with boracic acid, in the proportion of 1 to 8.

I generally scrape from the forearm with a long amputation knife; what I implant is a mixture of blood and epithelial cells, and looks like soft pink paste. In my early cases, I not only grafted at the time of the operation, but at subsequent dressings also.

Mr. C. E. WEST.

I think the meeting has been fortunate in having Mr. Marriage to introduce this discussion. The points of interest seem to be, firstly, those concerning technique; and secondly, the *pros* and *cons*. With regard to technique, it is best for each person to say what he does. To take the graft, I use any razor which is sharp; if I have any doubt about

it I shave myself with it on the morning of the operation: it is no good to trust to the instrument-maker to sharpen it. The Thiersch knife I consider a badly balanced and unsatisfactory implement. The next essential is to have a light hand. I do as thorough an operation as I know; I remove the posterior bony meatal wall as low as I dare. I do not make a point of exposing the dura mater, but I do not mind whether it is exposed or not. I clean the cavity out as far as I can by packing it with dry gauze, and I tampon it with gauze wrung out in 1 in 1,000 adrenalin. Then I cut the graft. I take it up with two fingers, and spread it out until it lies in contact with the wall of the cavity; then I tampon it into place with gauze wrung out in paroleine and bismuth subnitrate, which prevents putrefactive organisms multiplying, while the paroleine greatly aids the ease of extraction of the plug afterwards. I bring the ends out through the meatus, I form a flap, which I think is Ballance's original flap. It is most important to have an adequate opening; and if you keep a long, firm even curve in the incision, there is nothing lacking in æsthetic effect. It is necessary to pare the flap as thin as possible. It is a marvel to me how primary grafts take on a new raw bony surface. What I find most difficult is to get sufficiently close apposition between the graft and the relatively soft tissue of the deeper surface of the concha; it is there that I get most persistent granulations. With regard to the contra-indications, I agree with Mr. Marriage that one would not graft a confessedly infected labyrinth; and I will go much further. I used to think it was safe to graft a case of canal fistula in which I had good reason to suppose that the periosteum of the canal was intact; but I lost two cases from meningitis owing to doing that, and I have not done it since. And I do not now graft a case which has had undoubted symptoms of vertigo, whether inspection revealed a labyrinth lesion or not.

With reference to the pros and cons, in a routine case of chronic disease which will not get well without operation, I think one saves time, on the whole, by primary grafting, and in fewer cases are there troubles afterwards when this is done. I do not think the results brought forward are more than impressions, but I think one gets better hearing after grafting, as a rule, than without grafting. I think it is because in a proportion of the latter cases there is a filling up of the tympanum. We shall not form conclusions on this until we have an elaborate series of cases in which operation has been carried out under the same conditions, and there is a complete record of the hearing power before and after operation. I do not think it suffices to group

cases as "improved" and "not improved." I think that if you do the radical mastoid operation on a patient whose hearing was good before the operation, you will nearly always find his hearing is reduced afterwards. On the average, I think the hearing is not much altered either way. If you operate upon a patient who had pure middle-ear deafness, there is a chance of improved hearing after the performance of the mastoid operation. If there is not much cicatricial tissue in the tympanum, I do not think it matters much whether you have grafted or not.

I should like to hear the views of other members, particularly with regard to the dangers of skin-grafting in cases of possible infection of the labyrinth, a matter which impresses me more than anything else in regard to primary skin-graftings.

Mr. SYDNEY SCOTT.

Mr. Marriage's paper is a very interesting one, and impresses upon me the similarity of our experiences and views. I prefer, however, to transfer the graft by moulding it over gauze and packing it directly into the cavity, and have discontinued the use of paroline mentioned by Mr. West because of the possibility of a thin film intervening between the graft and bone and preventing intimate contact. Age presents no contra-indication, and when it is impossible to obtain a graft from a wasted infant the graft is taken from a more robust subject. Special treatment is necessary after skin-grafting on account of the formation of casts of epithelium which accumulate in the cavity afterwards. Will the President say whether this occurs after his method of grafting? [The PRESIDENT: Yes, it does, but I find that time puts it right.] Patients should attend regularly every three months, *whether they feel any complaint or not*, after the radical mastoid and skin-grafting operation, for the purpose of removing this epithelial accumulation, otherwise ulceration is apt to occur beneath. After a year or so the epithelial accumulations are much less troublesome. Like Mr. Marriage, I do not hesitate to apply a graft over a fistula of the external semicircular canal when the labyrinth is properly functioning, and I have never seen bad results from doing so. Where it is intended to drain the labyrinth for suppuration in it, I do not graft over the labyrinth, nor after opening the internal auditory meatus, as in the translabyrinthine operation; in these cases I confine the graft to the mastoid area. Favourable results can be obtained even when an extra-dural abscess has been opened by grafting after completing

the radical mastoid operation, but naturally one cannot employ the same technique in every case.

The President's method of grafting seems so simple that I intend to try it upon the next opportunity.

MR. MARK HOVELL.

Most of my cases have been treated by grafting, and therefore I cannot draw a comparison from experience between cases which have and those which have not been grafted. I think success greatly depends on the thinness of the graft. With regard to flattening the surface from which the graft is to be taken, I think the application of a paper-knife or some other straight edge draws the skin tighter and flatter than using the hand. With regard to accumulations of cerumen and skin, I agree that, as time goes on, the quantity lessens; they are universally present, and it is important that they should be removed.

MR. HUNTER TOD.

After the very excellent paper of Mr. Marriage, which has been so heartily supported by others, it is difficult to say anything against skin-grafting. And yet I would compare the two methods, grafting and non-grafting, for this reason—that I am one of those who in recent years have not made a practice of grafting.

Much depends on the character of the case. Probably those who have taken up grafting as a routine measure started with successes, because they did it first in simple cases. My experience has been among cases nearly all of which suffered from complications. At the London Hospital 10 per cent. of the mastoid operations have accompanying intracranial complications, and most of the patients are ill-nourished and are not brought to the hospital until they have extensive bone disease. I did not get good results from grafting in my early cases, partly owing to the class of case operated on, and partly, perhaps, owing to faulty technique on my part; therefore I largely gave it up. In the straightforward complete mastoid operation, without grafting, for chronic otorrhœa, for polypi and middle-ear disease without extensive bone disease, the average duration of time before healing takes place is two to three months; one expects the stitches to be removed within one week, and the patient to be out and about within three weeks. Mr. Marriage, I admit, has had quicker results, and I congratulate him on having 99 per cent. of his grafts take successfully.

My experience is that in a certain proportion of cases—certainly larger than Mr. Marriage mentioned—granulations spring up about three weeks after operation from points of underlying diseased bone which one thought had been removed.

Another group of cases is those in which there is general bone disease—mastoid osteomyelitis; these are very difficult to cure, either with or without a graft. One of my chief difficulties after the complete mastoid operation has been that instead of skin growing in, you get mucous membrane growing out from a patent Eustachian tube, sometimes spreading over the inner surface of the tympanic cavity and antrum. In such cases the patient may have good hearing, but there is still a mucous secretion. I ask whether Mr. Marriage finds this also, and whether he thinks that putting in a graft prevents this infection of the surface.

In operating I do not scrape the inner wall at all except around the Eustachian orifice, because the less you tamper with the inner wall of the promontory the less likelihood is there that scar tissue will form. If you leave the promontory alone you may find in some cases that for a long period it is lined with mucous membrane which gradually becomes normal, as in a "dry" perforation; but the hearing is much better than in those cases in which you get fine scar tissue growing up. So in a certain proportion of cases you benefit by not grafting. And if you can get skin to grow in normally over a bony surface, you are more certain of a permanent cure; normal skin will not grow where there is disease. If the skin grows in normally and completely lines the cavity within in eight or ten weeks, you may say that the patient is cured. Of course, our failures go to others; it is only the successful ones we keep. So I see cases of unsuccessful grafting, whereas my colleagues may see my unsuccessful cases of non-grafting. In a certain number of cases in which there is a small sclerosed mastoid, grafting seems to me to be quite unnecessary. Like Mr. West, I make a continuous cavity, removing the posterior wall of the meatus completely between the tympanic and mastoid cavities. I then fashion a Y-shaped posterior meatal flap, preserving the whole of the fibrous portion of the cartilaginous meatus, and if you cut it longitudinally a little below the median portion, you can throw this flap into the lower part of the mastoid cavity; this will often prevent the formation of granulation tissue between the tympanic cavity and the mastoid cavity, the area where, I think, trouble commences. I never make the concho-meatal flap, as it may cause a wide deformed meatal passage.

If you are certain that the graft will take at the primary operation, as Mr. Marriage asserts, I admit that probably grafting is better than non-grafting, because it shortens the case. But if the disease is limited, and there is a small cavity, you can do just as well without grafting, and often the hearing is even better. If the deafness is due to mechanical obstruction owing to the middle ear being filled with granulations, so that conversation is heard less than 12 ft. off, you can promise, if you do not over-curette the promontory, that on recovery ordinary conversation will be heard about 12 ft. off. But if you do a radical mastoid operation on a person who hears at more than 12 ft. (which should not often occur) the hearing is likely to be reduced afterwards. Formation of scar tissue about the stapes, whether the result of grafting or curetting, will considerably reduce the hearing.

Mr. J. S. FRASER.¹

Along with others I have found that there is not infrequently a tendency to drooping of the auricle after the radical mastoid operation. This is not only unsightly, but also narrows the external meatus, which was enlarged at the operation. In some cases the drooping is so marked that in order to inspect the cavity afterwards the surgeon must almost go down on his knees, or must tilt the patient's head over towards the opposite shoulder to an uncomfortable extent. To obviate this I have removed a crescentic piece of skin at the upper and posterior part of the curved retro-auricular incision. I always make this incision in the *hair margin* in chronic cases. The crescentic piece of skin removed is about 1 in. long and $\frac{1}{2}$ in. wide at its broadest part. For some time this small piece of skin was thrown away, but lately it occurred to me that the skin might be used as a graft to cover the facial spur. All will agree that there is a great tendency after the radical mastoid operation to the reformation of the posterior bony wall of the external meatus. No matter how freely this wall has been removed above so as to lay open the attic and aditus, and no matter how well the spur has been smoothed off below so as to expose the promontory and windows, within two or three weeks after the radical operation there is, in many cases, an exuberant growth of granulations from above and from below in the region of the cut edges of the posterior wall of the bony meatus. This exuberant growth is especially seen in cases in which the bone is very vascular or diploetic. In obstinate cases no method of after-treatment

¹ Communication read by Dr. Dan McKenzie.

seems to make much difference—packing or non-packing, spirit drops or boric insufflations, scarlet red or liquid paraffin. Only skin-grafting is of service, but to cut and apply a skin-graft from the inner side of the thigh takes up a considerable amount of time and probably inconveniences the patient for a period, though I cannot speak with any great authority on this subject. After trying for a time Ballance's method of skin-grafting ten to fourteen days after the radical operation, I came to the conclusion that it was not worth while. In any case, I have found that I cannot be troubled with carrying out this method.

The technique of the small procedure which I have devised is as follows: The crescentic piece of skin is outlined with the knife at the time of the original incision and, during the operation, is left attached to the periosteum covering the mastoid. When the operation is completed the cavity is syringed out with luke-warm saline lotion and temporarily packed with sterile gauze. The crescentic portion of skin is now excised with the knife or scissors and placed on the sterile towel covering the patient's head, so that the epidermic surface is next to the towel and the fatty subcutaneous surface upwards. The assistant now seizes one end of the graft with a pair of mouse-toothed forceps, while the operator does the same at the other end. In this way the graft is stretched between the two pairs of forceps. The subcutaneous fatty tissue is now picked up by the assistant with the aid of a third pair of forceps, while, with scissors curved on the flat, the surgeon dissects away the fatty tissue, leaving only the cutis vera and epidermis. The gauze is now removed from the mastoid wound and the skin-graft is applied to the cut surface of the facial spur and covered with an oblong piece of perforated oiled silk only slightly bigger than the graft itself. The skin-graft covered by the oiled silk is then held in position by the assistant, with the aid of a pair of angled forceps, while the surgeon packs the tympanic cavity, aditus and antrum with iodoform worsted, which retains the graft in position after the assistant has withdrawn his forceps. The retro-auricular wound is sewn up and dressings applied. If all goes well the wound is only dressed on the fifth day. Even when the iodoform worsted is removed the oiled silk remains in position and is taken out with angle forceps. The skin-graft invariably retains its position, and by the end of a period of ten days it is seen to be spreading out towards the antrum and also covering the floor of the meatus. In one or two recent cases healing of the cavity has been complete in six weeks. The whole procedure only adds at most five minutes to the time occupied by the radical operation.

Dr. DAN MCKENZIE.

I have followed out the ordinary Thiersch graft method, described by Mr. Ballance, for four years. But if anyone supposes that skin-grafting of any kind is going to turn the ordinary radical mastoid operation into a satisfactory operation he will be disappointed. It does not accomplish anything in the nature of magic; it does not transform what is a difficult and tedious operation into a satisfactory one. What it does do, however, is to shorten the period of convalescence. After grafting, the cavity is more rapidly covered with epithelium, and the ear dries up quicker. The time taken in grafting at the operation takes so little longer that those who have begun to graft continue to do so as a routine. The reason grafting has not become popular is that it is felt it is difficult to cut a graft. But in reality the difficulty is very much exaggerated. The chief necessity is a very sharp knife. With regard to subsequent hearing, that matter has been worked out by Welty, of San Francisco.

Dr. DUNDAS GRANT.

I have not made a routine practice of grafting, but have done it in many cases. I have never regretted having grafted, but have frequently regretted not having done so. With regard to hearing, I am convinced, in company with Sir William Milligan, that the best results are seen after grafting. I had a good proof of this in a case in which both ears had to be treated for a similar condition, one with a graft, and the other, for certain reasons, without a graft. The hearing on the side grafted was very much better than on the other. It is difficult at first to believe that a primary graft placed on bare bone will take root and stay there, but it is now accepted that primary grafting is an excellent course to pursue. In some of the cases one sees now—for instance, soldiers—the patients have very coarse skins, and from them it is not easy to get a nice thin graft. The graft should be cut very quickly.

There are cases in which Nature creates the equivalent of a graft, as in those in which, through long neglect, a cholesteatoma has formed, with a preservative matrix. It is a mistake to take that away and put another in its place. Ten years ago I brought a boy to the Otological Society in whom I had found such a cholesteatoma. A year afterwards I brought him up again to show how well Nature's graft had acted. It was dry at the end of a month, and skin was lining the

cavity. A few weeks ago I was asked to see a soldier who had been under my care; it was this same boy, now developed into a strong Scottish sergeant. He came because of deafness *in the other ear* owing to the bursting of a shell. The cholesteatoma-matrix had stood the hardships of months of campaigning, and was as well as if perfect skin-grafting had been done. Usually, of course, cases are now seen before a cholesteatoma has had time to develop. There are also cases in which the cavity is very small and grafting is scarcely called for. There is, on the whole, no method more perfect than the use of the Thiersch graft, as formulated by Mr. Ballance, and in the "primary" form as now described by Mr. Marriage.

Mr. W. STUART-LOW.

After personal experience I would say I am not in favour of grafting. I used it for some time, but I got as good results without it if I was careful. I think surgeons do not always take sufficient care in regard to a number of points respecting the mastoid operation. First, in regard to the preparation of the patient, I insist on tobacco and alcohol being stopped, and I give the patients a rest from work. I operate on the throat by enucleating the tonsils and removing the adenoids; a resection of the nasal septum is also done, if necessary, and consequently the patient is properly prepared for the operation. It is also important to remove bad teeth. I cut a large flap into the concha, and then there is a good deal of epithelium to lay down. This is laid down over the facial ridge, catgut stitches being put in to maintain the flap in position. Do not curette the inner wall of the tympanic cavity on any account, otherwise you will get scar tissue, and the hearing will not be good. I now use the blood serum treatment. Having removed the diseased substance and dried the cavity, I put in 10 c.c. of horse serum, and the cavity is packed with gauze and a dressing applied. To avoid contraction of the meatus I employ a cage, and a bandage over the cage. I have never had any contraction of the meatus since using this. Forty-eight hours afterwards the plug is taken out, and no more after-plugging is ever required. If the epithelium is not coming on quickly enough, I blow into the cavity dry sterile powdered pig-skin epidermis.

Mr. MARRIAGE (in reply).

I have been very interested in hearing the views of different members, but I do not seem to have much to reply to. In the treatment of these cases, as regards the small details of technique, it is

largely a matter of what one is used to, but as regards method the question is, Which one will give both patient and surgeon least inconvenience? From that point of view, I think there is no question that primary grafting saves the patient much pain and gets him well quickly, and saves the surgeon much trouble too. In the patient I showed this afternoon, healing occurred in five weeks, and I have only seen her three times since the operation; all the treatment she has had was done by the ordinary dresser at the hospital. That is a big contrast to the cases which we used to treat by plugging, or even with scarlet red; cases then required much more attention than that.

In answer to Mr. Tod, one cannot guarantee that the Eustachian tube will be closed, and that there will not be some extension of mucous membrane. But by not scraping the inner wall of the tympanum at all, he is much more liable to get trouble than if he had scraped. Still, even grafting will not always close the Eustachian tube, and I have never suggested that grafting will make all cases heal immediately. I think Mr. West is too pessimistic about putting grafts over a bony fistula. I have done many such cases, but up till now I have not had meningitis developing in consequence.

I was glad to hear of Mr. Scott's method of transferring the graft: I have not myself tried it. I started with a section lifter, and got accustomed to that, and I do not think there is much difficulty. I was interested to hear from Mr. Scott in conversation that he had had a case of primary grafting of the mastoid which healed up in one week, and another in ten days. I do not profess to equal that, but I see that it can happen, because so often when you take out the first dressing, the whole cavity is practically covered, especially the bony cavity. The chief difficulty is the posterior meatal wall.

Mr. Fraser's method, cutting out a crescentic piece, seemed from the description to be more complicated than cutting a straightforward graft. I would rather cut a graft and cover the whole cavity than put in a layer of skin and tuck it away in the manner described.

I do not agree with Mr. Stuart-Low, that after grafting you get a contraction of the meatus; one of the advantages of grafting is that you do not get that contraction. Getting the skin to heal over the raw surface, at any rate in the upper part of the meatus, prevents it from contracting up, and I think Mr. Stuart-Low must have been unlucky in the way he cut his flap, or in his cases, if he has had many cases of contraction of the meatus.

Otological Section.

President—Dr. ALBERT A. GRAY.

(February 18, 1916).

Case of Tumour on Concha after Radical Mastoid Operation (shown at the last Meeting).¹

By W. M. MOLLISON, M.C.

THE tumour on the concha proved to be skin, with some fibrous tissue underneath; there was no cartilage in it.

DISCUSSION.

THE PRESIDENT: Is it keloid? It does not yet present that appearance. One views these growths which appear in or near a scar with suspicion lest ultimately they may become malignant, however innocent they appear to be. When there is a recurrence, the second growth is more rapid than the original one.

Sir WILLIAM MILLIGAN: Is the tumour keloid in structure? The importance of the case is from the practical point of view. My experience is that if one removes a keloid it returns, and generally becomes worse than before. Keloid in the neighbourhood of the ear is a disappointing condition with which to have to deal.

Mr. HERBERT TILLEY: Some twenty years ago a patient of mine had a very extensive keloid after the mastoid operation, and the question arose many times in the subsequent years whether it should be removed. Ten days ago, however, it was impossible to see any keloid in that patient, but its place was taken by a widened scar, as opposed to the usual narrow linear scar. I think it is wiser not to operate on keloid. As an alternative treatment I would suggest radium; we have had many cases of keloid at the

¹ See *Proc. Roy. Soc. Med.* (Otol. Sect.), ix, p. 6.

Radium Institute, and I know of no pathological condition for which radium is more successful than in a case of keloid.

Mr. MOLLISON (in reply): For a keloid it had a very narrow base; one could not tell whether it started in the scar or not; probably it must have done so. Re-operation has since been performed on the mastoid, and the meatus is already tending to contract so much that gauze cannot be got into the cavity¹; I do not know whether this may be due to the same cause as the "keloid."

(February 18, 1916.)

Audible Tinnitus in a Boy (shown at the last Meeting).²

By W. M. MOLLISON, M.C.

SINCE he was here the boy has been operated upon for removal of his tonsils and adenoids, but the tinnitus continues. His tubes have been inflated without causing any alteration in his tinnitus.

DISCUSSION.

Dr. H. J. BANKS DAVIS: At the last meeting I saw Mr. Lake compress the carotid and stop the noise; but when the pressure was withdrawn the sound returned, which seems to point to a vascular condition as the cause. It would be interesting to ascertain whether the noise stops when the boy is under an anæsthetic. That would settle the point as to whether it is a "habit" or not.

Mr. E. D. DAVIS: Two months ago I had a case in a Belgian, who produced the noise by contraction of the temporal muscles. I taxed him with it, and told him that it was of no importance.

Mr. CLAYTON FOX: On nipping the boy's nostrils and making him blow out his cheeks to fix the soft palate, the noise ceased, so possibly it was caused by the contraction of the tensor palati.

Dr. DUNDAS GRANT: I had a similar case, in which the noise ceased when I passed a Eustachian catheter and bougie. I think the noise was due to sudden separation of the glutinous walls of the Eustachian tube. It remained absent for some minutes, and then recurred. I think this should be treated as a nerve condition, being, in my opinion, of the nature of a habit spasm.

¹ Note.—March 18: The cavity was completely dry.

² See *Proc. Roy. Soc. Med.* (Otol. Sect.) ix, p. 7.

The PRESIDENT: If the case is of the nature of *tic convulsif* the outlook, according to some authorities, is not satisfactory, for such cases are said ultimately to develop mental trouble.

Mr. SYDNEY SCOTT: Last September I saw a patient (Miss M.), with normal hearing, who appeared to have voluntary control over the tensor tympani muscle. She was a lady with an extraordinarily neurotic family history and was personally extremely introspective. I could hear a sound through the auscultatory tube which corresponded with the patient's effort to move the drum of the ear. I was able to exclude jaw movements, and could actually see a change in the direction of the light reflex from the membrane which indicated movement. During the apparently increased tension of the membrane, the lowest tones on the Bezold-Edelmann series were inaudible. At first the control was experienced in the right ear only, but after practice the patient was able to produce the same effect in the left ear. The examination was made with eyes closed, and the signs were repeatedly consistent with the patient's statement of her sensations.

Mr. MOLLISON (in reply): My assistant and I have looked at the palate repeatedly, but could not see a trace of movement. The noise stops momentarily when the patient opens his mouth wide. I see no reason why it should not be caused by the contraction of the tensor tympani.

(February 18, 1916.)

Sections through the Footpiece of Stapes from the Case of Otosclerosis shown at the last Meeting.¹

By A. A. GRAY, M.D. (President).

THE sections are from four cases of otosclerosis of one year's, three years', twenty-five years', and sixty years' duration.

In the first of these the change in the bone was different from the ordinary otosclerotic changes; there was only absorption of bone, no deposit of it, so that the diseased parts scarcely stained at all. All that was left were a few blood-vessels and broken-down bone cells. The patient had fairly advanced phthisis, which may account for the absence of deposit of bone.

The second case was also the subject of phthisis, but it was not advanced. Here the new-formed bone stained deeply, and was sharply defined; there was no fixation of the stapes.

¹ See *Proc. Roy. Soc. Med. (Otol. Sect.)*, ix, p. 7.

In the third case the stapes was united by bone from the anterior margin to the oval window. The bridge of bone which united the stapes to the oval window was exceedingly fine. It was not one-tenth part of the diameter of the cross-section of the footplate of the stapes.

The fourth case was that of an old lady, aged 80. Rarefaction in one portion of the wall of the labyrinth had gone so far that there was practically no bone left. Osteoclasts were absorbing the bone. Manasse was of opinion that the bone is not absorbed by osteoclasts but by simple pressure.

An interesting feature in all the cases is the position of the anterior line of demarcation. It was almost identical in all of them, despite the great differences in the duration of the disease. I think it is a mistake to suppose that the bony changes are necessarily progressive. There was no sign of inflammatory activity in any of the sections. There was no evidence of change in the organ of Corti, nor in the ganglion spirale, except for the ordinary post-mortem changes. I am satisfied, however, that there is a change in the nerve structures, but our methods are not sufficiently refined to enable us to detect them.

Dr. DUNDAS GRANT: Sometimes the clinical conditions and the tuning-fork responses are not typical, and yet the osteoporotic changes are found post mortem. I should like to know whether Gellé's test was tried, and whether there was raising of the lower limit of audition.

February 18, 1916.)

Histofogical Preparations of the Labyrinths and Tympana of a Soldier suffering from Deafness due to a Bullet Wound of the Vertex of the Skull.

By SYDNEY SCOTT, F.R.C.S.

C. T., AGED 42, a private in the Lincolnshire Regiment, was wounded on November 13 near Ypres, and died in France from meningitis on December 14, 1914. The autopsy showed the fracture was confined to the vertex of the skull. The histological examination reveals extensive intra-tympanic hæmorrhage on the right side. The serial sections demonstrate that the tympanic membrane, the footplate of the stapes, the annular ligament, and the membrana secundaria are perfectly intact and there is no hæmorrhage within the labyrinths.

Clinical notes: The patient was admitted into the base hospital three days after being wounded; he was able to walk, was quite rational, but very deaf. He remembered being wounded, and stated he was standing in a trench at the time; when hit he fell down, and noticed loud buzzing noises in the ears immediately, with deafness. There was a lineal suppurating wound of the scalp $3\frac{1}{2}$ in. long over the right parietal bone, running from the lambda to the parietal eminence. The X-rays showed comminution and fissured fracture of the skull beneath the wound. There was no paralysis of the face or limbs, and the superficial and deep reflexes were normal.

Ears: Some cerumen was present in both ears, but no blood or clot or cerebrospinal fluid. The tympanic membranes were normal, but the light reflex on the right side was dark blue, an appearance which suggested the probable presence of blood in the tympanic cavity.

For several days after admission he could only hear shouting close to his ear, then he improved somewhat and could hear an ordinary clear voice about a yard away. He always spoke loudly, as if he could not hear his own voice.

		Right ear		Left ear
Edelmann-Galton whistle,	...	2.5 x 0.8 mm.	...	4.5 x 0.8 mm.
No. 473		= 19,000 d. v.		= 13,920 d. v.
Tuning-fork, 256 d. v.	...	Rinne neutral	...	Rinne negative
		Schwabach - 6 sec.		Schwabach - 2 sec.
Watch normally heard at	...	Just audible close to but not touching		
3 or 4 ft.		either auricle		

Unfortunately, owing to the lack of instruments, we do not know what the low tone-limit was.

A fortnight after admission the patient became hemiplegic on the left side. The skull was explored, and fragments of the fractured parietal bone removed. A small tear in the dura mater was discovered, and some bone splinters were removed from the superior parietal lobule between the angular gyrus and the great longitudinal fissure. The hemiplegia was not relieved, and the patient succumbed to meningitis a fortnight later.

The necropsy showed extensive softening of the right cerebral hemisphere. The pia-arachnoid over the external and mesial surfaces of the right hemisphere were infiltrated with pus, which was limited to this region. There was no basal meningitis, and no fracture of the temporal bones or base of the skull. The temporal bones were fixed in formalin and decalcified with nitric acid, and imbedded in celloidin in the

ordinary way. The sections, which are arranged in series, were stained with iron hæmatoxylin, and show extensive blood-clot throughout the right tympanum, and only traces of blood-clot in the left tympanum.

DISCUSSION.

The PRESIDENT: We have to thank Mr. Scott for showing us those sections, beautiful as his always are. Why is it that the hæmorrhage came into the tympanum without any sign of injury in the deeper parts?

Sir WILLIAM MILLIGAN: I should like to ask Mr. Scott why this patient was not operated upon a fortnight earlier? I should have thought, from a general surgical point of view, immediate operation was called for; he ran every risk of having septic meningitis of his vertex. In regard to the deafness, I cannot understand how a patient who has a hæmorrhage into one tympanum and an intact labyrinth could be so deaf as the tests show. I ask whether there has not been some disturbance in the interior of the cranium which accounted for the profound deafness; whether the cortical centres for hearing were not injured by a commotio cerebri?

Dr. DUNDAS GRANT: In support of what Sir William Milligan has said, the tuning-fork on the mastoid was diminished to -6 sec. on one side and -2 sec. on the other. In the right ear the Galton whistle was heard at a higher pitch than in the left (apparently normal) ear. I therefore think there is justification for supposing there was some disturbance of the auditory centres. I have had at least one case in which deafness followed shell concussion, with similar hæmorrhages on the surface of the membrane. There were hæmorrhagic bullæ, which discharged into the meatus, from which came a discharge of blood. In some of the septic cases there has been previous traumatic rupture of the membrane, and infection has followed on account of the circumstances in which the patient was placed.

Mr. MOLLISON: In answer to Sir William Milligan, surely it is not necessary in the case of a man who has had a head injury to suppose he has necessarily anything wrong with his cortical areas because he has a high degree of deafness? This man had been exposed to shell shock, and had had a bullet wound of the scalp. I myself—and, no doubt, Sir William also—have seen men in whom complete deafness has resulted for a time from a wound in the head which had no relation whatever to the ear. Recently I saw an officer who was shot through from the right temporal region to the left malar bone; he was completely blind and deaf. But during three or four weeks his blindness gradually passed off and his hearing was restored; he could hear an ordinary conversation without trouble. These sections are of great interest, as showing there is nothing wrong with the labyrinth; but it is unfortunate that they do not shed any light on the causation of cases of deafness from shell shock.

Dr. KELSON: The interest of the case is increased by the fact that there is no fracture of the base of the skull. Probably Mr. Scott has noticed that many patients who have been shot about the face or jaw speak often spontaneously of a discharge from the ear which commenced after the injury, and has persisted ever since. This occurs too often to be a mere coincidence; I think we must consider these as injuries due to indirect violence.

Sir WILLIAM MILLIGAN: I was not referring to the psychic cases of deafness in which there had been no injury to the skull. I have seen many cases of profound deafness due to shell shock, and have found the most severe deafness in men who had been buried for the time being.

Mr. J. F. O'MALLEY: The tests show the deafness here to be of the middle-ear type, although emphasis has been laid by some speakers on it having been a nervous injury. It is not due to shell shock. With regard to the deafness sometimes mentioned by soldiers who have had wounds of various parts of the head, my experience has been that many of them have evidence of old ear lesions. I have notes of several who manifestly have had loss of tympanic membrane, dating, perhaps, from a serious lesion in early childhood, and although the deafness preceded the injury, it only assumed importance afterwards.

Mr. SYDNEY SCOTT (in reply): The records of this case are too fragmentary to show what was the cause of the bilateral deafness. The damage to the right cerebral hemisphere should be taken into account. The chief feature of the case is that an injury to the vertex of the skull was associated with hæmorrhage into one middle ear without any fracture of the base of the skull, or of any injury to the tympanic membrane, labyrinth, membrana secundaria or stapes. The source of the hæmorrhage cannot be found in any of the sections (of which there are about 200 in series on each side). Considering all the circumstances of the case, it is doubtful what would have been the result of an earlier intervention.

(February 18, 1916.)

Removal of a Piece of Shell from the Cerebellum of a Soldier wounded at Ypres.

By Sir WILLIAM MILLIGAN, M.D.

THE piece of shell (specimen shown) passed through the auricle, cut a groove in the soft tissues, and entered the anterior portion of the left cerebellar lobe. Severe hæmorrhage resulted, possibly from the lateral sinus. When seen a suppurating sinus led down to the foreign body. Removal was easily effected and complete recovery ensued.

(February 18, 1916.)

Case of Tuberculous (?) Septic (?) Meningitis following Chronic Suppurative Otitis Media; Operation; Repeated Lumbar Punctures; Prolonged Illness; Recovery.

By Sir WILLIAM MILLIGAN, M.D.

C. I., MALE, aged 27, admitted to the Manchester Royal Infirmary (Throat and Ear Department) on December 28, 1914, complaining of discharge from left ear, violent headache, and vomiting.

Previous history: Had severe attack of broncho-pneumonia as a boy, followed by pulmonary tuberculosis. In February, 1912, had a second attack of right-sided pneumonia, followed by extensive breaking down of the lung and formation of a large apical cavity. Enormous quantities of secretion were expectorated. Shortly before this second attack of pneumonia the left middle ear had become infected and had discharged freely. In November, 1914, he joined the R.A.M.C.; he was sent to Southport and was vaccinated there. Was very much upset by vaccination (?), violent pain in the head and profuse discharge from the ear. Was sent home. Discharge from ear profuse, violent headache, vomiting, dull, lethargic condition, gradually deepening into semi-unconsciousness. Admitted to Royal Infirmary on December 28, 1914. Temperature 99° F., pulse 80, respirations 23. Left-sided facial paralysis, pupils unequal.

December 29: Complete post-aural operation and decompression over temporo-sphenoidal and cerebellar areas.

December 30: Evening temperature, 104° F.

December 31: Mid-day temperature, 99.5° F.; evening temperature, 102° F. Retraction of head; severe headache. Kernig's sign present; patient delirious, very noisy; swore immoderately when touched. Lumbar puncture performed: fluid under pressure; very turbid. Report *re* cerebrospinal fluid: "The fluid contains pus; no tubercle bacilli or other organisms found. Culture yields no growth of organisms. The condition may therefore be tuberculous. (Signed), Loveday." Ice-bags applied to head. Urotropine, 20 gr., every four hours.

During the following ten days repeated lumbar punctures, fluid still turbid.

January 11: Condition critical; saline enemata with brandy.

January 14: Temperature came down from 102° F. to 98.4° F.; pulse from 138 to 120, and respirations from 36 to 24.

Temperature remained about normal until January 19, when it went up to 101° F., pulse 112, respirations 32. Repeated lumbar punctures. From this date a gradual improvement set in.

February 7: Morning and evening temperature normal; patient rational.

February 22: Patient allowed out of bed.

March 8: Wound healed; complete epidermization of antro-tympanic cavity.

March 16: Discharged.

Patient has remained well; his mental condition good; pulmonary symptoms not so marked.

Attention is specially drawn to the value of repeated lumbar puncture.

DISCUSSION.

Mr. J. F. O'MALLEY: I would add a third query to the two propounded in the title—namely, as to the possibility of cerebrospinal meningitis. I have now had a case for several weeks, and the patient was under my care before the present attack, as three months ago I removed an aural polypus from him. He had a rash. The cerebrospinal fluid was examined the second day of the disease, and though pus was found there were no organisms after three days' incubation. This was so until the eighth day, when no organisms were found by direct examination by two bacteriologists, but there were two colonies of meningococci on culture. The early symptoms of Sir William's case and mine were almost identical, except that my patient did not vomit, but had headache all the time. The medical officer at the isolation ward was very expert, and felt confident about the diagnosis, though it was suggested by the bacteriologist that the rash might be septic. As the cerebrospinal fluid finding was so often negative it was thought it might be otitic meningitis. Lumbar puncture is performed on alternate days, and the patient is recovering.

Dr. H. J. BANKS DAVIS: I do not think a patient who appears to have been so ill could live with cerebrospinal meningitis from December 31 to March 16, and the history is against this diagnosis. As a rule, when extremely ill, the patients succumb to cerebrospinal meningitis within a week or ten days.

Mr. SYDNEY SCOTT: Was there any evidence to show whether the infection was from the ear, and if so was it by the translabyrinthine path? I recollect a child who died of meningococcal meningitis after pneumonia, from whose tympanic cavities two kinds of organisms (on each side) were isolated—namely, intracellular cocci and capsulated extracellular cocci, the first corresponding to the meningococcus which had been isolated from the spinal fluid during life, and the second resembling pneumococci.

The PRESIDENT: I presume the pus from the case was not injected into a guinea-pig, as that might have solved the problem as to whether it was a case of tuberculosis?

Sir WILLIAM MILLIGAN (in reply): It never occurred to me that the case might be cerebrospinal meningitis; I regarded it as an ordinary septic case, and expected that an organism would be found. Only two bacteriological examinations were made, and no organisms were found. Still, I should be rather sceptical of its being cerebrospinal meningitis; there was no rash. The other day a case was sent in with symptoms of meningitis, and I diagnosed septic meningitis; but when the fluid was reported on by the bacteriologist, he said he had discovered a single meningococcus. I expressed my doubt as to that being sufficient to support a diagnosis of cerebrospinal meningitis, and it proved later to be an ordinary septic case. I do not know what the path of infection in this case was: it was not the labyrinth; it was a pure middle-ear case. What ought we to consider septic meningitis? Is the presence of this established without demonstrating organisms? I have been in the habit of rejecting the diagnosis unless organisms are found. With regard to injection of material into a guinea-pig, I still do that occasionally, but did not do so in this case. I do not think the decompression did him much good; what I regarded as most beneficial were the repeated lumbar punctures which kept the intracranial pressure down, thus preventing death from compression of his own vessels and respiratory paralysis.

(February 18, 1916.)

Malignant Disease of Middle Ear following Chronic Suppurative Otitis Media; Extension to Mastoid Cells; Operation; Rapid Recurrence; Employment of Radium; Temporary Relief.

By Sir WILLIAM MILLIGAN, M.D.

J. B., MALE, aged 58, seen on April 3, 1913, suffering from right-sided suppurative middle-ear disease of many years' duration. Right ear discharging freely; perforation of posterior segment of membrana tympani; polypus; slight tinnitus; no vertigo.

April 7, 1913: Polypus removed; chromic acid applied.

June 5, 1913: Ear quite dry; perforation not healed.

July 8, 1914: Recurrence of polypus—rather vascular.

July 21, 1914: Removal of polypus; malignancy suspected, but not proved microscopically.

August 6, 1914: Discharge slight, but foetid; slight hæmorrhage. Antiseptic treatment ordered.

March 8, 1915: Radical mastoid operation; granulation tissue very vascular.

April, 1915: Antro-tympanic cavity unhealed; definite microscopic evidence of malignancy.

May, 1915: Post-aural cicatrix broken down; fungating granulation tissue; slight pain; no glandular involvement.

July, 1915: Patient losing weight: several fairly severe attacks of hæmorrhage from ear; slight enlargement of glands; severe pain.

August, 1915: Radium emanation tube (25 mg. element) inserted into fungating mass.

September, 1915: Pain and hæmorrhage much relieved; growth shrinking; free discharge from the meatus.

November, 1915: Growth half its former size; recurrence of pain; glandular involvement greater than before; facial paralysis. Radium emanation tube (25 mg. element) again inserted.

December, 1915: Local relief, but systemic decline; evidence of toxæmia.

January, 1916: Patient much weaker; no great alteration in local condition.

February, 1916: Patient rapidly losing ground; glandular involvement about the same; no increase of local growth, but progressive loss of strength and weight. Condition hopeless.

DISCUSSION.

The PRESIDENT: I think cancer arises more frequently in, and is more common in, a suppurating middle ear than in an ear which is not so affected. I saw a case in which a growth was deep in the meatus; there was no suppuration, and it was secondary to malignant disease in the breast. Radium was employed, and the growth completely disappeared and did not return; but the patient died of a lesion in the lung, secondary to the breast cancer.

Sir WILLIAM MILLIGAN (in reply): With regard to a remark made by a member as to radium increasing pain, my experience has been the opposite—namely, that it relieves pain. It also stops the hæmorrhage, which is a very important point. I do not know who made the report on radium to which reference has been made. Its action in cases of sarcoma is extremely beneficial, but in carcinoma its benefit is doubtful. However, we are only just beginning to understand about the action of radium, which is generally used only in the cases which are otherwise hopeless; it has never had a proper chance in cases which we speak of as operable. To condemn radium at the present stage is absurd. Radiologists seem to agree that embedding is the best way in which to use it.

(February 18, 1916.)

A Note upon the Significance of Unilateral Sero-mucous Catarrh of the Middle Ear in Persons over Middle Age.

By Sir WILLIAM MILLIGAN, M.D.

UNILATERAL sero-mucous catarrh of the middle ear in persons over middle age should always be regarded with suspicion. A diagnosis of simple catarrh is often made and is just as often wrong. Some incipient malignant process or syphilitic manifestation should always be thought of, and a guarded prognosis given.

In many cases malignant ulceration of the naso-pharyngeal mucosa or a malignant growth of the basi-sphenoid is discovered if careful rhinoscopic examination is made, or some syphilitic process is found to be the underlying factor in the case.

A careful rhinoscopic examination by mirror or naso-pharyngoscope should be made as a matter of routine in all such cases, as this frequently leads to important and far-reaching clinical discoveries.

DISCUSSION.

Mr. TILLEY: I wish to support emphatically what Sir William Milligan has said. In the last fifteen years I have seen eleven cases of endothelioma of the nasopharynx; and one of the saddest of these I saw three years ago. The patient had seen two well-known aural surgeons in this country, and three on the Continent. He complained of deafness, which varied in degree, and he was told it was catarrhal deafness. One surgeon catheterized him every week for two months. I saw him rather late in his illness, when one nostril was blocked, and there was a bluish, congested-looking mass in the nasopharynx. It eventually invaded his skull, and he died of cerebral symptoms and in great pain. In these cases when the deafness has been present for a matter of weeks you generally find some difficulty in the movement of the levator palati on the same side; this is due to mechanical impairment rather than to a nerve lesion. Then follows anæsthesia of the second division of the fifth nerve, evidenced by loss of sensation on the outside of the lower jaw and over the lower portion of the cheek. This anæsthesia and the weakness of the levator palati are almost pathognomonic of the condition. If in such a case 100 mg. of radium are embedded for twelve hours, it is extraordinary how the condition will disappear. Last week I saw a man in whom the growth was as large as the end of one's thumb, and the patient could not blow down the right nasal fossa. Radium was embedded a month ago, and when I saw my patient last week no trace of the growth was to be seen; the nasal breathing was free, and the pain in the temporal region had disappeared.

Dr. DAN MCKENZIE: Possibly the reason why the case which Sir William Milligan referred to was overlooked by those who saw the patient earlier was that the growth was not then sufficiently large to be visible by the post-rhinoscopic mirror, therefore, all cases of deafness should be examined with the naso-pharyngoscope as a matter of routine. Such a case as Sir William Milligan relates would not be missed by this means, even in the earliest stage.

Dr. JOBSON HORNE: I have always practised and taught a routine examination of the post-nasal space and of the posterior nares with a mirror wherever it is possible, in all cases of diseases of the ear, the nose, or the throat. The importance of that practice and of that teaching has been fully established not only by the clinical surprises from time to time observed and by the aids to diagnosis afforded, but also by the life-long calamities at times thereby prevented.

Mr. SYDNEY SCOTT: It is sometimes difficult to decide whether some cases of middle-ear catarrh are secondary to malignant disease or to syphilis. Last November I saw a patient at the National Hospital for Paralysis, who for six months previously had been treated at another hospital for unilateral middle ear catarrh and Eustachian obstruction. He then developed optic neuritis and paralysis of the left sixth and seventh cranial nerves. The left eighth cranial nerve was unaffected. In the nasopharynx was an ulcer which involved the infundibulum of the left Eustachian tube; it looked like malignant disease, but the nerve lesions pointed to syphilis. (I shall refer to this case more fully another time.) More recently I saw another patient whom I had examined three weeks previously, and found nerve-deafness in the right ear. During the interval he had awakened with loud tinnitus and great deafness in the left ear, which had been previously normal. I found the signs of nerve-deafness with great loss of bone-conduction and of high and low tones, but the left Eustachian tube was impervious even to bougies. His history of syphilitic infection was very definite; he had been treated intramuscularly with salvarsan two years ago, followed by a course of mercury for one and a half years. A week later I found the Eustachian obstruction had gone, the membrane returned to the normal, but the hearing was still worse.

(February 18, 1916.)

Cases illustrating Otogenic Facial Paralysis.

By DAN MCKENZIE, M.D.

Case I: Post-operative Paralysis.—Male, aged 29, shown before this Section on October 12, 1912, after recovery from lateral sinus thrombosis and serous meningitis, for which translabyrinthine drainage

had been adopted. The patient had two operations in all. At the first a huge cholesteatomatous cavity was disclosed, in the depths of which the facial nerve was found slung across the space like a thread and devoid of all bony protection from the genu to the stylo-mastoid foramen. But no paralysis was observed until several hours after the second operation, at which the labyrinth was penetrated to reach the internal auditory meatus, so that the injury to the nerve must have been inflicted during this procedure. The course of the paralysis since its inception illustrates the striking tendency to recovery, but often only to partial recovery, manifested after severe injuries to the nerve in the temporal bone. For six months no change could be detected in the face, but at the end of that period a little fibrillary movement in the fibres of the orbicularis palpebrarum was perceived when the patient was made to close the eyes tight, and the improvement has gone on continuously, but very slowly indeed, during the three years that have elapsed since then. Now he can close the eye, the ala nasi shows movement, and the naso-labial fold appears when he smiles. But none of these movements is equal to that corresponding to it on the other side of the face. The patient himself is of opinion that the improvement is still continuing, and with this I agree, but progress is extremely slow. The reaction of degeneration has never been present.

Case II.—Male, aged 32, shown before this Section on May 14, 1915, as a case of recovery after purulent meningitis. In this, as in the first case, translabyrinthine drainage was effected, but the paralysis was observed on the patient recovering from the anæsthetic. The first sign of recovery was seen on December 17, 1915, thirteen months after the operation, in slight movement of the orbicularis palpebrarum, the dilator naris, and the levator anguli oris. The improvement, in this case, unlike the last, is proceeding rapidly.

Case III: Concussion (?) Paralysis.—This case is included in the series, as it seems to suggest the explanation of facial paralysis following mastoid operation in which no sign of exposure of the nerve at the operation has been detected. A male, aged 25, received what was supposed to be a shrapnel wound of the mastoid on May 1, 1915. He was rendered unconscious for a couple of hours, but no facial paralysis was noticed until two days later, and in four weeks the paralysis began to get well. There is now no sign of it. A small fragment of metal which had lodged in the tympanum was removed by the exhibitor eight weeks ago, by means of a radical mastoid

operation, and the route of the missile was then seen to pass through the mastoid and the posterior meatal wall close to the tympanum, and within a few millimetres of the Fallopian canal. As far as could be made out the nerve itself had not been exposed.

Case IV: Paralysis from Otitis Media Purulenta.—Woman, aged 32, who came to hospital with chronic suppuration in the left ear and complete facial paralysis of the corresponding side of the face of one week's duration. The radical mastoid operation revealed cholesteatomatous disease, which had exposed the facial nerve for about $\frac{1}{2}$ in. in the tympanum. The facial paralysis began to improve six months after the operation, but in this case, as in the first, complete recovery delays its coming.

Case V: Herpetic Paralysis.—Male, aged 32. This case was published *in extenso* as one of herpes zoster oticus combined with recurrent laryngeal paralysis in the *Journal of Laryngology* (September, 1915, p. 339). It is now shown here with the facial paralysis nearly well. (The laryngeal paralysis lasted a few days only.)

Case VI: Paralysis from Otitis Media Catarrhalis.—Boy, aged 8. After an attack of acute pain in the right ear, facial paralysis suddenly appeared. There has been no discharge from the meatus. When first seen on December 8, 1915, the membrana tympani showed some dull redness in the upper hemisphere, but it did not rupture. There was no herpes. Consequently this is evidently one of those rare instances of paralysis from simple catarrh of the middle ear, several cases of which have been shown before this Section. The paralysis is rapidly recovering, having been complete for a month only.

The most important cases of facial paralysis from the surgeon's point of view are, of course, those which follow mastoid operations. In my experience, severe facial paralysis from this cause is very rare, and when it does occur it nearly always undergoes great improvement, if not complete recovery, sooner or later. Years, indeed, may elapse—according to Vohsen as many as ten years—and yet recovery take place. I suggest that the reason for recovery, even after complete section of the nerve, lies in the fact that section of the facial nerve, passing as it does through a fine canal in the bone, may occur without the ends of the nerves being separated from each other, and as the intervening gutter or bridge of bone may be assumed to serve as a guide

to the sprouting nerve-fibres, their ultimate union is probable. On the other hand, as the cases now exhibited show, complete recovery may be delayed for a very long time; indeed, it may never be reached. This, also, I ascribe to the narrow channel through which the nerve passes. If the nerve is severed the scar tissue which forms will partially block the passage and prevent the full reunion of the nerve.

I have said that in my experience, and, I believe, in that of all otologists, complete facial paralysis from operative trauma is extremely rare. On the other hand, I find paresis relatively common. This paresis varies in severity. In the mildest cases a slight lagging behind, or delay in closing the corresponding eyelid as compared with the movement of the opposite eyelid in the ordinary action of the involuntary winking of the eyes, is all that appears, and it is so slight as to escape all except the most jealous observation, and so transient that it is visible for but two or three days. Then there are the more obvious and familiar cases, in which the paresis slowly spreads from the eyes to the rest of the face, and after four or five weeks slowly disappears. In many of those cases the surgeon is prepared for the paralysis by the appearance of a facial twitch at the operation. In others, again, no warning has been given, and the paralysis comes as a surprise. It is in such cases that I suggest concussion or contusion to be the cause of the nerve lesion—by contusion I mean, rather, hæmorrhage into the canal, the effects of which are not immediately apparent after the operation. Case III illustrates this type, and I can recall another in which the paralysis followed the use at the operation of a blunt set of chisels.

When the nerve is exposed by natural dehiscence in its bony envelope, or by caries of the bone, post-operative paralysis, when it appears, is probably due to septic or traumatic neuritis. It appears about three days subsequent to the operation, lasts about six weeks, and finally disappears.

We frequently come across cases in which a twitch during the operation reveals the exposure of the nerve, but in which, for some unknown reason, no paresis or paralysis ensues.

In the radical mastoid operation one great difficulty we have to contend with is to clear disease from the posterior or postero-external wall of the tympanum (the sinus tympani), where caries often lurks. Curetting here is liable to injure the nerve, and yet to neglect this corner is to run the risk of the disease being prolonged and the benefit of the operation being lost.

DISCUSSION.

The PRESIDENT: This question of facial paralysis is a very practical one. I have had my share of cases of post-operative facial palsy. I had one case in which the nerve had been exposed by cholesteatoma, and the operation was the ordinary radical one for mastoid disease. In these cases I have found that the quicker the suppuration ceases, the quicker is the repair of the nerve. That is what one would expect on a priori grounds.

Dr. H. J. BANKS DAVIS: In the first case the note says: "The reaction of degeneration has never been present"; I suppose that this is an error and means that it has always been and is still present; I think it is there in every case of paralysis after the first week or ten days following injury to the nerve below the nucleus. It may, of course, only be "partial reaction of degeneration" and not complete, but the responses to electrical stimulation cannot be normal if facial paralysis is present, unless it be "functional."

Dr. DUNDAS GRANT: I wish to support what Dr. McKenzie says with regard to the jamming of the nerve by cicatricial tissue in the tube. I would remind you of a paper which was read by Ferdinand Alt at the International Congress in London, 1913. It was on old-standing cases of facial paralysis which he had operated upon by deliberately opening the Fallopian canal, and liberating the facial nerve from pressure. I should feel some diffidence in doing this, but there may be others who have experiences of the kind to offer. I agree it is very rare for the paralysis to remain complete for any great length of time. I think, with Dr. Gray, that recovery is hastened by using the continuous current. I should like to hear what results have been obtained by the anastomosis of the facial nerve with one of the other nerves; I have not had the opportunity of seeing the ultimate results of that procedure; operators do not seem anxious to record them. In opening the aditus I use a guard, and I think it has helped me to avoid facial paralysis. But the guard must be held by an experienced person, otherwise it may defeat its object, especially if there is anything like dehiscence in the canal. Anatomical investigations have shown that the course of the facial nerve varies; in some cases it runs vertically downwards from the inner wall of the aditus; in others it runs for some distance horizontally outwards, and then makes an almost right-angled turn downwards; and in the latter cases it is specially liable to injury. By careful operating one can chip off the corner of the bend without cutting through the facial nerve. In using the chisel, I find it best to turn the bevel of it over, so that the tendency is for the tool to work towards the surface rather than the deeper parts. I ask whether it is common for the facial nerve to be affected when translabyrinthine drainage is carried out. It is such a fascinating procedure that one would like to be assured it is free from that risk. Of course, a freer drainage can be secured by making an opening in another place, though the ensuring of asepsis is then probably more difficult.

Sir WILLIAM MILLIGAN : If Dr. McKenzie will do me the honour of reading Milligan and Wingrave on "Diseases of the Ear," he will find a chapter devoted to facial nerve paralysis. I rise, however, to draw attention to the injury which is often done by curetting the middle ear. If it is to be done at all, it should be carried out from behind forwards; it is then less risky. If, unfortunately, one divides the nerve in the course of an operation, one should have the courage to open up the aqueduct immediately as far as possible, and endeavour to twist a little piece of foil or india-rubber tissue round the nerve, so as to conduct it to the distal opening in the bone, and so encourage repair. I have seen two cases of bilateral facial paralysis in middle-ear disease. I agree that there is generally some recovery in these cases, and the lapse of years does not make the case hopeless. It is rare to find a patient suffering from absolute and permanent palsy of the nerve. We in this country do not see anything like so many cases of this kind as are to be met with in many Continental clinics.

Dr. JOHNSON HORNE : I agree with what has been said about the importance of care in curetting the middle ear in the mastoid operation, and apart from any mastoid operation I would urge particularly the advisability of confining all curettage of the middle ear to patients indoors and in bed. Referring to Dr. Grant's point about reversing the chisel when removing the bridge in the mastoid operation so that the bevel of the chisel is on the under surface, at that stage of the mastoid operation I work with a curved chisel with the bevel on the under surface. With the use of that instrument and Hartmann's punch forceps I have always escaped injuring the facial nerve. Apart from traumatism, there have been cases of ear disease in which facial paralysis was inevitable, such as tuberculous disease of the ear.

Mr. SYDNEY SCOTT : We do not find that facial paralysis always follows the translabyrinthine operation for meningitis,¹ although it occurred on the third day in the first case which Mr. West and I described. One should be specially careful to keep close to the floor of the internal auditory canal.

Dr. DAN MCKENZIE (in reply) : I will not reply at length at this hour, but suggest the subject for a discussion at another session. Unless you curette the ear very thoroughly in some cases, you will not get it to heal up. And in the cases of caries in the posterior part of the tympanic cavity, the spot where the nerve is most liable to be exposed and where facial paralysis is produced, one must always curette there if the case is to be cured. I think we should decline to remove a polypus from the middle ear except through the mastoid; I have twice seen a polypus attached to the exposed trunk of the nerve, and if it had been removed in the ordinary way, facial paralysis would have been inevitable.

¹ *Proc. Roy. Soc. Med.*, 1908-09, ii (Otol. Sect.), p. 11.

Otological Section.

President—Dr. ALBERT A. GRAY

(May 19, 1916.)

The Pathology of Otosclerosis, Congenital Syphilitic Deafness, and Paralabyrinthitis.

By J. S. FRASER, M.B., F.R.C.S.Ed.

OTOSCLEROSIS.

At the present time it appears to be impossible to state with confidence that the condition variously known as "otosclerosis," "dry middle-ear catarrh," "spongification of the labyrinth capsule," "otitis vasculosa petrosa," "otitis stapedio-vestibularis," "petrous osteodystrophy," &c., is a pathological and clinical entity. It may be that the condition we know as "otosclerosis" is made up of two or more pathological conditions in somewhat the same way that "oedema" may result from affections of the heart, kidneys, nerves, or from inflammatory processes. All we know is that, apart from suppurative otitis media and adhesive processes in the tympanum, there are many cases of chronic progressive deafness (usually accompanied by tinnitus) in which functional examination of the auditory apparatus gives us the results we associate with a lesion of the sound-conducting mechanism—i.e., raised lower tone limit, lengthened bone-conduction and negative Rinne test. Microscopic examination of such specimens as have been available have shown certain changes in the labyrinth capsule, especially in the anterior bony margin of the oval window. In the early stages of the disease we

¹ From the Ear and Throat Department, Royal Infirmary, Edinburgh, under the charge of A. Logan Turner, M.D., F.R.C.S.E.; and the Laboratory of the Royal College of Physicians, Edinburgh, under the charge of Professor James Ritchie.

find here vascular spongy bone containing numerous spaces which show a central blood-vessel surrounded by some connective tissue. Osteoclasts are not found at this stage. Later on the diseased area becomes sclerosed by deposit of new bone in the walls of the spaces. The process usually extends to the stapedio-vestibular joint and results in bony fixation of the stapes. In the early stages the nerve structures are normal as a rule, but in the later there is usually a considerable degree of atrophy affecting Corti's organ, the stria vascularis, the spiral ganglion and the cochlear nerve.

THEORIES AS TO THE PATHOLOGY OF OTOSCLEROSIS.

There appear to be only four possible ways in which the affection may arise: It may be congenital; it may follow inflammatory changes in the middle ear; it may be due to infection through the blood; it may be caused by abnormal conditions in the nerve supply of the parts affected. For convenience we may take these possibilities in the following order:—

(1) Otosclerosis is regarded by some as a chronic inflammatory process spreading from the mucoperiosteum of the middle ear and following attacks of catarrhal or suppurative otitis media. The inflammatory condition is supposed to linger about the niche of the oval window and to invade the bone from the deep layer of the mucosa. Such an invasion may be favoured by the anastomosis which occurs at this spot between the tympanic vessels and those of the bony labyrinth capsule. Further, as Walker and I have pointed out, the joints in the middle ear are the only articulations in the body covered by mucous membrane and therefore liable to infection from the surface.

This view of the pathology of otosclerosis does not by any means exclude the hereditary transmission of a tendency to the disease. It may well be that in certain families the mucosa of the middle ear and the bony capsule of the labyrinth are congenitally weak, and therefore unable to resist infection from the surface. In the same way there seems to be evidence that in certain families cases of suppurative otitis media are specially liable to develop labyrinthine or intracranial complications. In cases of middle-ear suppuration several observers have noted changes in the bony capsule of the labyrinth similar to those seen in the early stages of otosclerosis—i.e., stage of *ostitis vasculosa* or *spongification*.

Against the theory that otosclerosis is an inflammatory condition

following otitis media we must place the facts that—(a) in most cases the mucosa of the middle ear is normal (it is well known, however, that otitis media may pass off and leave little or no trace); (b) plasma cells are absent (I am not sufficiently skilled in pathology to deal with this point, but it has been stated that plasma cells are not invariably present in inflammatory conditions); (c) areas of osteoporosis are found in spots far distant from the mucosa of the middle ear (this will be dealt with later).

(2) According to the second theory, otosclerosis is a congenital anomaly of the process of growth in the petrous temporal bone, due to the presence of certain determinants in the patient's blood. This anomaly only becomes manifest after puberty. Under normal conditions the growth of the membranous labyrinth and its bony capsule ceases soon after birth, though islands of cartilage may occur in the labyrinth capsule in the region of the anterior margin of the oval window (figs. 9 and 10). According to the second theory growth continues in this cartilage bone and later spreads to the footplate of the stapes, finally resulting in ankylosis. Those who hold this view lay great stress on the hereditary transmission of the disease, though they admit that the immediate cause of otosclerosis may be provided by puberty, by the puerperium, or by disease of the tympanic mucosa. Further, Siebenmann, who supports this theory, points out the similarity of the changes seen in otosclerosis to those which occur in a rib in cases of empyema. (In such cases the process must surely invade the rib from the periosteum?)

(3) A third group of otologists look on otosclerosis as a primary disease of the bone, the infection coming by way of the blood-stream, as in osteomalacia, rheumatism, &c. Ferreri regards otosclerosis as closely allied to osteomalacia, and finds that almost all patients suffering from the latter disease are also the subjects of otosclerosis. A variant of this view is held by our President, Dr. A. A. Gray, who has put forward the theory that otosclerosis is an aseptic necrosis of certain areas in the labyrinth capsule—apparently as a result of aseptic infarction. Gray believes that similar areas occur in other bones of the skeleton. It would certainly be of great interest to examine other bones, though it would not be so easy in them to identify areas of otitis vasculosa as it is in the dense labyrinth capsule. A grave objection to the theory that otosclerosis is a puerperal infection is the fact that it is very common in young unmarried women, while the cases so commonly met with in men must, of course, be explained in another way. If, for a

moment, we consider theories (1) and (3) together, we may compare them with those put forward regarding the connexion between tonsillitis and rheumatism. Formerly it was held that attacks of tonsillitis were of "rheumatic origin"—i.e., that the tonsil was presumably infected through the blood-stream. More recently opinion has turned round, so that it is now held that rheumatism is of "tonsillar origin"—that is to say, that the blood-stream becomes infected from the tonsils. In my opinion the number of cases of otosclerosis which are looked on as *secondary* to otitis media is likely in the future to grow larger, while the number of the so-called *primary* cases is likely to decrease.

(4) Lastly, otosclerosis is regarded by a small group of observers as due to a trophic disturbance—i.e., as a degenerative atrophic process. According to this theory the changes (sometimes) found in the ganglia and nerves are primary, while those in the labyrinth capsule are secondary. In this connexion it may be noted that certain cases, which clinically appeared to be examples of *nerve deafness*, on microscopic examination showed spongification of the labyrinth capsule with (Siebenmann), or without (Bruehl), ankylosis of the stapes. This type of otosclerosis associated with nerve deafness—the so-called "atypical otosclerosis"—is markedly hereditary. Again, spongification of the bone in front of the oval window has been observed in an old woman aged 82 (Wolff), and in two other cases in old people (Mayer). On the other hand, advanced cases of otosclerosis have been microscopically examined, in which no degenerative changes were found in the nerve structures.

NATURE OF THE CHANGES IN THE BONE IN OTOSCLEROSIS.

According to Siebenmann the changes are as follow: (1) Vascular and fibrous marrow penetrates the normal Haversian canals and dilates them. (2) Next there is lacunar resorption by giant cells of the cartilage bone of the labyrinth capsule, resulting in the formation of large spaces containing blood-vessels and numerous cells. (3) New deeply staining bone is deposited in the walls of these large spaces (the bone takes on the basic hæmatoxylin stain). (4) The new bone becomes dense and gradually loses its affinity for the basic stain.

Manasse describes the changes thus: (1) Granulation and osteoid tissue are formed in the pre-existing spaces in the bone. This is the primary active process. The original bone of the labyrinth capsule disappears without absorption by giant cells. (2) The spongy new bone

is destroyed by canaliculization and lacunar resorption by osteoclasts; new marrow spaces are thus produced. (3) Bone is again formed by osteoblasts in the new marrow spaces; this bone becomes more and more compact by diminution in size of the marrow spaces. In this way new lamellar systems are formed, but the new compact bone is sharply marked off from the normal bone of the labyrinth capsule.

CASE OF CHRONIC MIDDLE EAR SUPPURATION AND CHOLESTEATOMA COMBINED WITH OTITIS VASCULOSA (OTOSCLEROSIS) OF THE LABYRINTH CAPSULE. (Figs. 1 to 6 inclusive.)

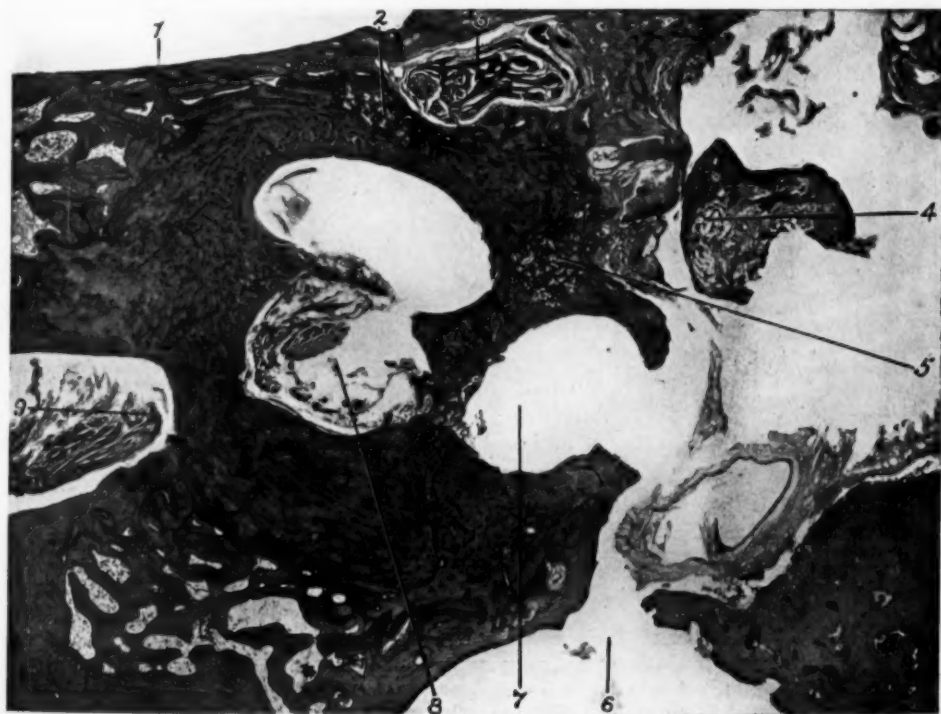


FIG. 1 (Section 385).

Vertical transverse section through the region of the anterior margin of the oval window, showing the area of spongification (5). The untidy condition of the section is explained in the text. 1, floor of middle fossa; 2, area of otitis vasculosa; 3, facial nerve; 4, ossicle (incus?) showing otitis vasculosa; 5, area of otitis vasculosa in anterior margin of oval window (seat of election); 6, jugular bulb; 7, basal coil (the bony spinal lamina and membranous structures have been removed—artefact); 8 and 9, internal meatus, with nerves. ($\times 8$.)

All writers on the pathology of otosclerosis take it for granted that the more deeply staining bone seen in the walls of the vascular spaces in the early stage of the disease is new-formed bone. In my experience, however, new-formed bone is usually pale and stains well with eosin, but not with basic stains. Such new bone may be seen in the walls of the mastoid air cells from cases of subacute mastoiditis.

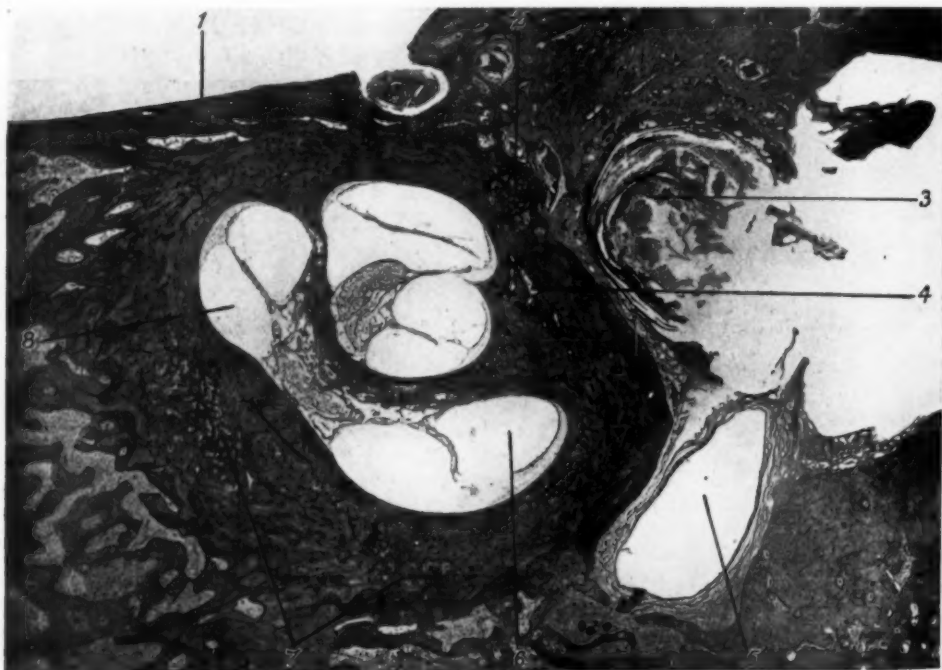


FIG. 2 (Section 295).

Vertical transverse section through cochlea, showing three areas of otitis vasculosa (otosclerosis). 1, middle fossa; 2, area of otitis vasculosa; 3, cholesteatoma; 4, remnant of lymph space in capsule of cochlea; 5, tubal part of tympanic cavity—the tympanic cavity in this case is divided into an upper part (lined by cholesteatoma) and a lower part communicating with the Eustachian tube; 6, scala vestibuli of basal coil; 7, two areas of otitis vasculosa; 8, scala tympani of basal coil. ($\times 8$.)

Accordingly, it seems probable that the bone which stains deeply with hæmalum or hæmatoxylin is not new bone at all, but merely the old bone of the region, the staining reaction of which has been altered

by the change it has undergone owing to the vascular dilatation and the consequent increased supply of lymph. If we grant that in the stage of osteoporosis some irritant, inflammatory or toxic condition be present, it will be obvious that in order to combat this condition the part needs an increased blood supply. The vessels of the Haversian spaces therefore dilate, the marrow proliferates, and the spaces become enlarged. It is not necessary that the bone should be removed by osteoclasts.

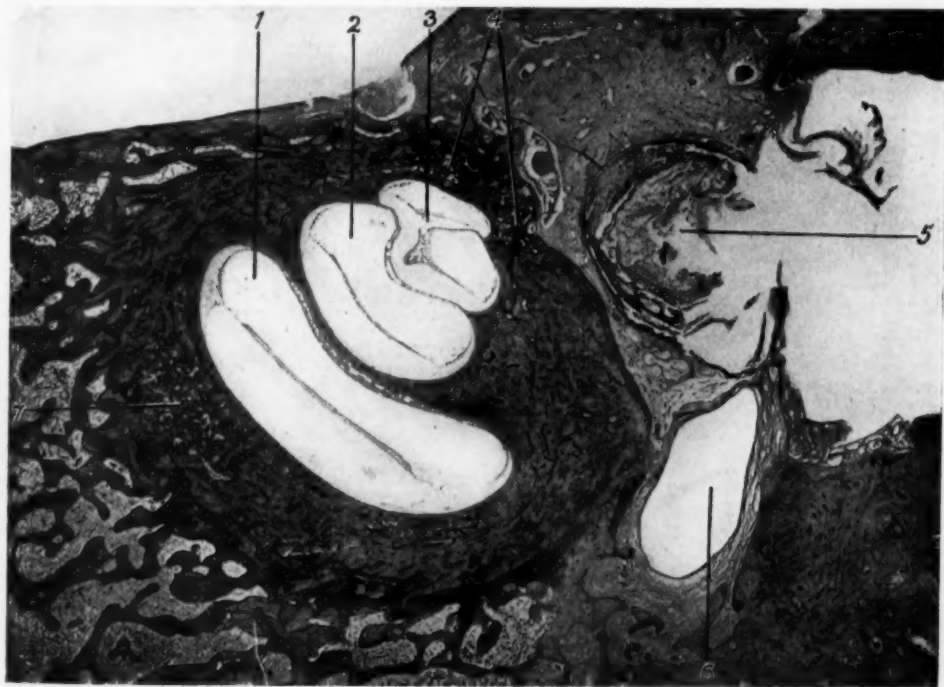


FIG. 3 (Section 255).

Vertical transverse section through cochlea showing two areas of otitis vasculosa. At the apex of the cochlea the bony changes reach the endosteum. 1, basal coil; 2, middle coil; 3, helicotrema; 4, two small areas of otitis vasculosa; 5, cholesteatoma; 6, tubal part of tympanic cavity; 7, area of otitis vasculosa. ($\times 8$.)

Bone yields before long-continued pressure exerted by structures much softer than itself—e.g., the enlarged mastoid antrum and tympanic attic in cases of cholesteatoma, in the walls of which I have never observed

osteoclasts. Later on, when the local chronic inflammatory or toxic process has been overcome, the vascular supply diminishes and the bone again becomes sclerotic and loses its affinity for basic stains.

In support of the first-mentioned view as to the pathology of otosclerosis—namely, that it is a chronic inflammatory process invading the labyrinth capsule from the deep layer of the mucosa in the region

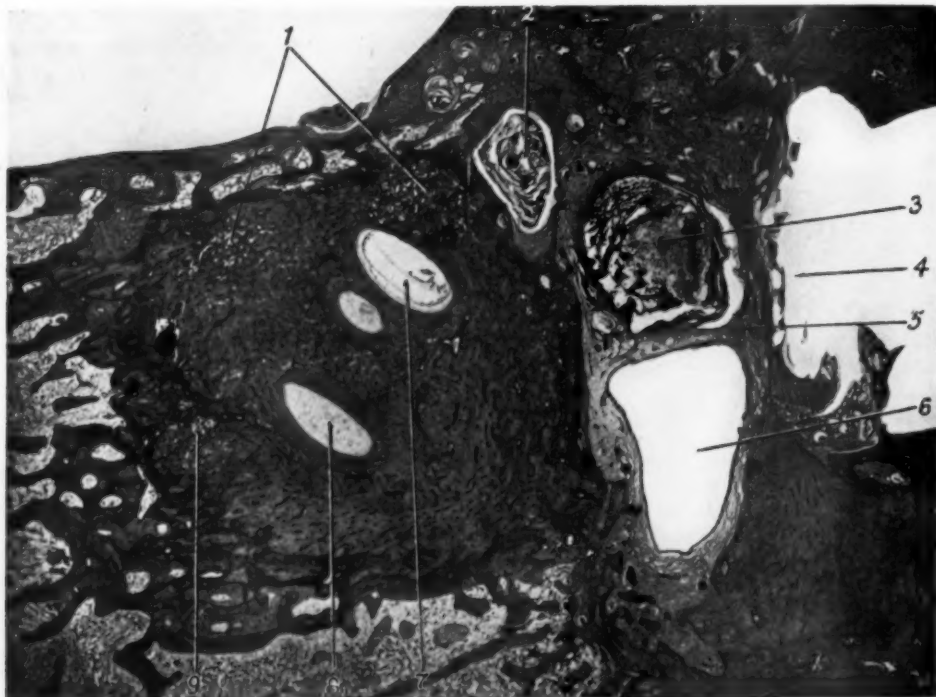


FIG. 4 (Section 195).

Vertical transverse section through cochlea showing three areas of otitis vasculosa in the cochlear capsule. 1, areas of otosclerosis; 2, tensor tympani; 3, cholesteatoma; 4, external meatus; 5, tympanic membrane; 6, lower part of tympanic cavity; 7, apical coil of cochlea; 8, basal coil of cochlea; 9, area of otitis vasculosa. ($\times 8$.)

of the anterior margin of the oval window—I bring forward the following case, the clinical notes of which are, unfortunately, very meagre:—

J. H., male, aged 19, had suffered from chronic middle-ear suppuration of unknown origin for many years. The patient was a foundling, and nothing is known as to his parentage; he was a very dull boy and "backward" in every way according to the account of his guardian. He was in and out of the poorhouse on several occasions, before his last admission on May 11, 1911.

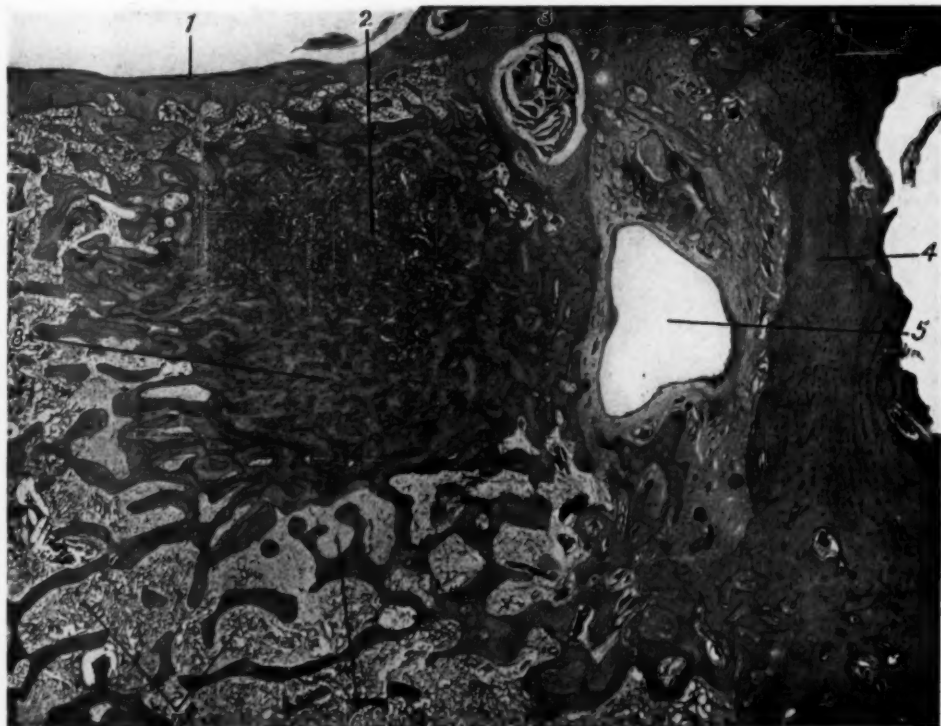


FIG. 5 (Section 135).

Vertical transverse section through capsule of cochlea, showing a large area of otitis vasculosa in front of the hollow spaces of the cochlea. 1, middle fossa; 2, large area of otitis vasculosa; 3, tensor tympani; 4, anterior bony wall of external meatus; 5, tubal part of the tympanic cavity; 6, fatty marrow; 7, fibrous marrow; 8, normal cartilage bone (interglobular bone) of cochlear capsule. The section is made anterior to the hollow spaces of the cochlea, and only shows the cochlear capsule. ($\times 8$)

Death occurred on May 15, and at the autopsy a large temporo-sphenoidal abscess was discovered. As the patient had not been seen by an otologist, and as no functional examination had been carried out, it was not at first



FIG. 6 (Section 445).

Vertical section through the posterior part of the labyrinth in the region of the semi-circular canal. Note the spongy condition of the bone (2) on the inner wall of the mastoid antrum (4). 1, superior canal; 2, area of *ostitis vasculosa*? passing in towards fossa sub-arcuata; 3, floor of middle fossa; 4, cholesteatoma in antrum; 5, external canal; 6, posterior canal towards the ampullary end—the canal here contains bone dust (artefact); 7, saccus endolymphaticus; 8, crus commune; 9, posterior cranial fossa. Note change in the bone (*ostitis vasculosa*) passing inwards from the cholesteatoma matrix on inner wall of antrum. In this situation the veins from the inner wall of the antrum pass through the fossa sub-arcuata to the posterior cranial fossa to join the inferior petrosal sinus. The condition seen in the photomicrograph is, however, not found under normal conditions. ($\times 8$.)

DEVELOPMENT OF THE LABYRINTH CAPSULE STUDIED IN RELATION TO THE PATHOLOGY OF OTOSCLEROSIS. (Figs. 7 to 11 inclusive.)

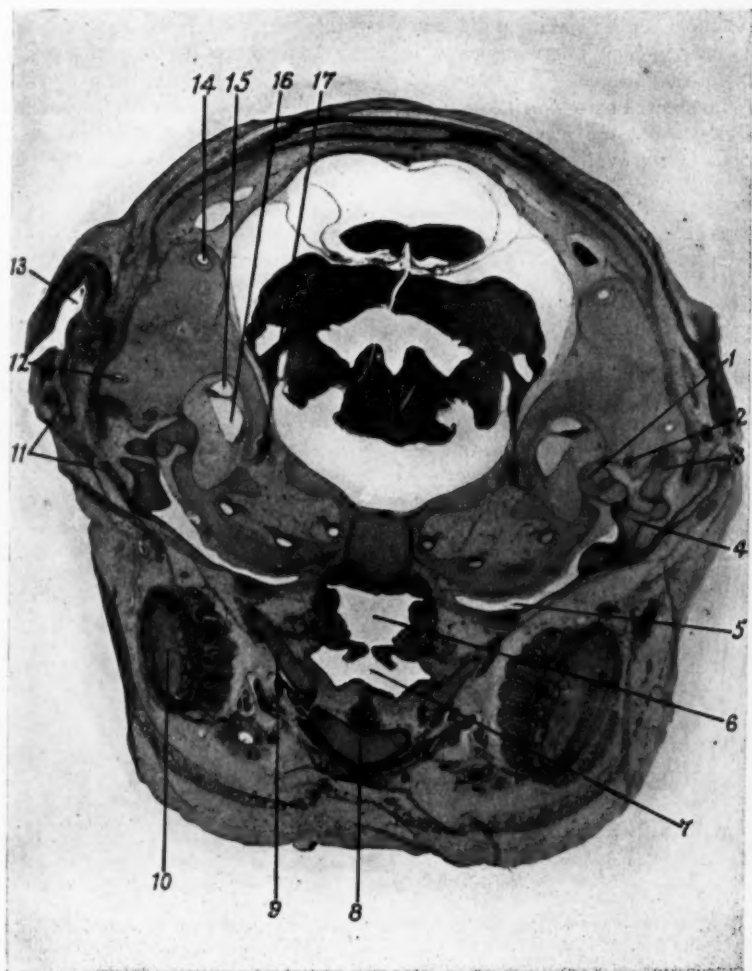


FIG. 7.

Vertical transverse section through head of foetal pig showing the foetal cartilaginous capsule of the labyrinth on both sides. The endolymph spaces are patent, but the perilymph spaces are still filled with foetal connective tissue. 1, footplate of stapes; 2, facial nerve; 3, incus; 4, malleus; 5, Eustachian tube; 6, naso-pharynx; 7, mouth cavity; 8, hyoid; 9, Meckel's cartilage; 10, lower jaw; 11, external auditory meatus; 12, external canal; 13, hollow of auricle; 14, superior canal; 15, utricle; 16, sacculle; 17, cochlear nerve. Note the cartilage capsule of the labyrinth extending from above 14 to the basi sphenoid which lies above 6. ($\times 8$.)

thought to be worth while to examine the ear microscopically. A vertical saw-cut was therefore made through the temporal bone from the external to the internal meatus in order to demonstrate cholesteatoma in the tympanic cavity and attic. As the specimen did not show this very well, it was determined to examine the ear microscopically in order to observe the condition of the labyrinth (cholesteatoma deafness?). In view of the fact that the labyrinth capsule showed extensive areas of *ostitis vasculosa*, it may readily be imagined that I was not too pleased with myself for having destroyed with the saw the regions of the oval and round windows. It is further unlucky that the plane of the saw-cut is not a favourable one for microscopic examination of the

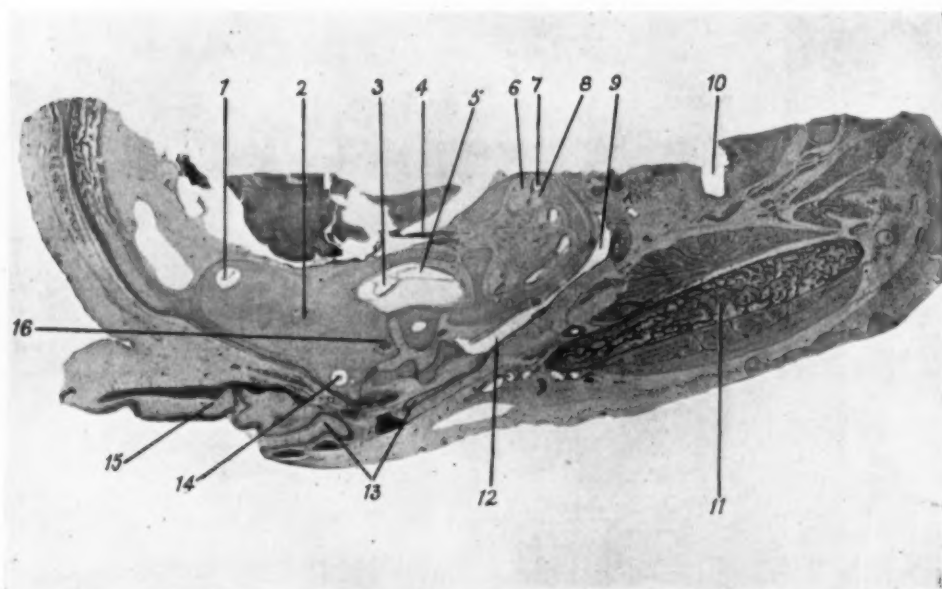


FIG. 8.

Vertical transverse section through head of fetal pig at later stage than preceding photomicrograph (No. 7). The section corresponds to the left half of fig. 7, but lies horizontally instead of vertically. The perilymph spaces of the vestibule and canals are now patent, but in the cochlea the perilymph space is still filled by fetal connective tissue. 1, superior canal; 2, vessels of fossa subarcuata; 3, utricle; 4, cochlear nerve; 5, saccule; 6, scala tympani still solid; 7, cochlear canal; 8, scala vestibuli still solid; 9, Eustachian tube; 10, mouth cavity; 11, lower jaw; 12, tympanic cavity only reaches malleus and anterior margin of oval window; 13, external auditory meatus; 14, external semicircular canal. 15, auricle; 16, facial nerve. The posterior and upper parts of the tympanic cavity are still filled with fetal connective tissue. The perilymph space of the vestibule and canals is now patent, though that of the cochlea is still filled with fetal connective tissue. ($\times 8$.)

labyrinth. The results of the examination may be summarized as follows: The Eustachian tube showed normal epithelium, but the submucous tissue was thickened and showed cystic spaces (fig. 4). The tympanic cavity, aditus and antrum were lined by cholesteatoma (figs. 3 and 6). The bony walls of the attic and antrum show large spaces containing connective tissue and blood-vessels, but no giant cells (ostitis vasculosa?) (fig. 6). On the inner wall of the tympanum in the anterior margin of the oval window there is an area of

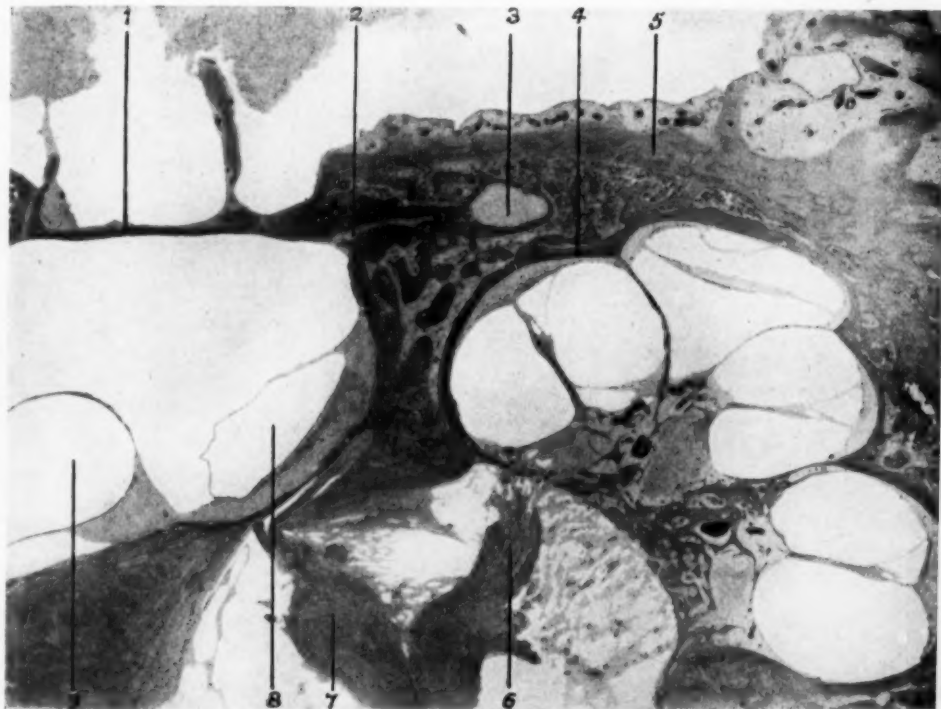


FIG. 9.

Horizontal section through labyrinth of full term fetus showing island of cartilage (3) in the bone of the anterior margin of the oval window. 1, footplate of stapes; 2, track of cartilage; 3, foetal rest of cartilage; 4, endosteal bone; 5, lamellar bone; 6, cochlear nerve; 7, vestibular nerve; 8, sacculus; 9, utriculus. ($\times 12$.)

ostitis vasculosa in the labyrinth capsule (fig. 1). If we start at this point we can trace the spread of the disease in the wall of the labyrinth between the periosteal and cartilage bone round to the region of the internal auditory

meatus, and over the apex of the cochlea to the anterior part of the bony capsule of this structure (figs. 2 to 5). At the apical coil of the cochlea the spongification reaches the perilymph space (fig. 3). Further back on the inner wall of the aditus and antrum, in the region where the vessels of the fossa subarcuata begin, the bone shows large spaces containing blood-vessels and connective tissue (fig. 6). The marrow surrounding the labyrinth capsule appears at all parts to be very fibrous (figs. 3 and 4). Beyond this the marrow is fatty, but cellular marrow is entirely absent.

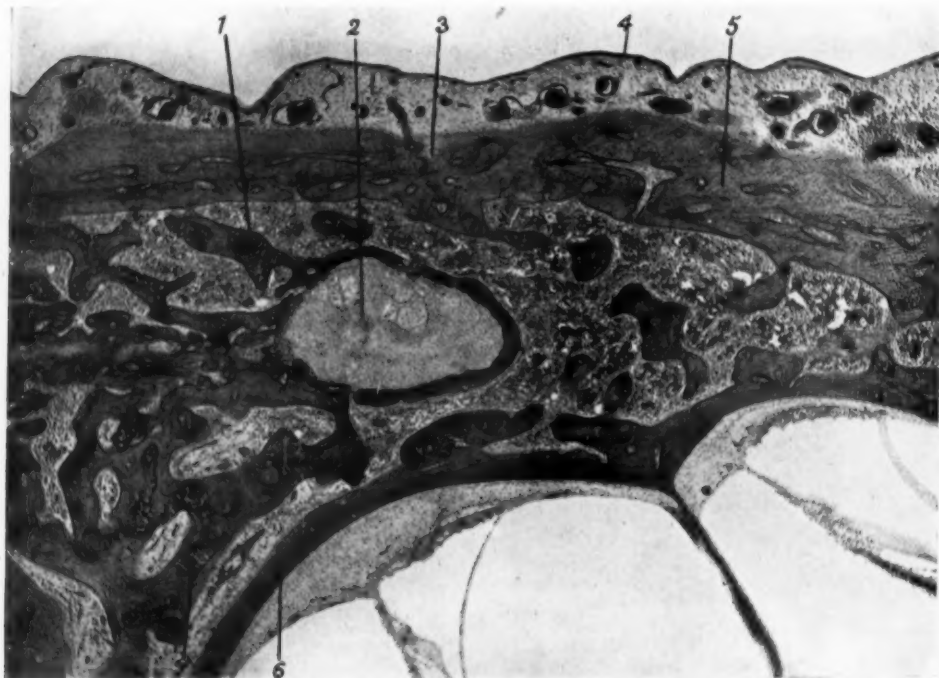


FIG. 10.

Part of fig. 9. Anterior margin of oval window from full term fetus showing remains of fetal cartilage. 1, cartilage or interglobular bone; 2, rest of fetal cartilage; 3, lamellar or periosteal bone; 4, mucosa of tympanum; 5, lamellar or periosteal bone; 6, endosteal bone; 7, cartilage or interglobular bone. ($\times 45$.)

In the case of specimens obtained from the human subject it is never wise to be dogmatic in regard to changes in the delicate nerve structures of the labyrinth, even in cases (such as the present) where the temporal bone was

obtained within twenty-four hours of death and at once placed in fixative. With this proviso it may be stated that Corti's organ appears to be atrophied in all coils; only the outline of this structure is visible. The cells of the spiral ganglion also appear to be shrunken, and their place is taken by connective tissue. The vestibule, along with the ampullary ends of the canals, were destroyed by the saw-cut.

I have found changes, somewhat similar to those seen in the labyrinth capsule of this case, in the malleus and incus obtained from cases of chronic middle-ear suppuration in which the ossicles were diseased (figs. 13 and 15).

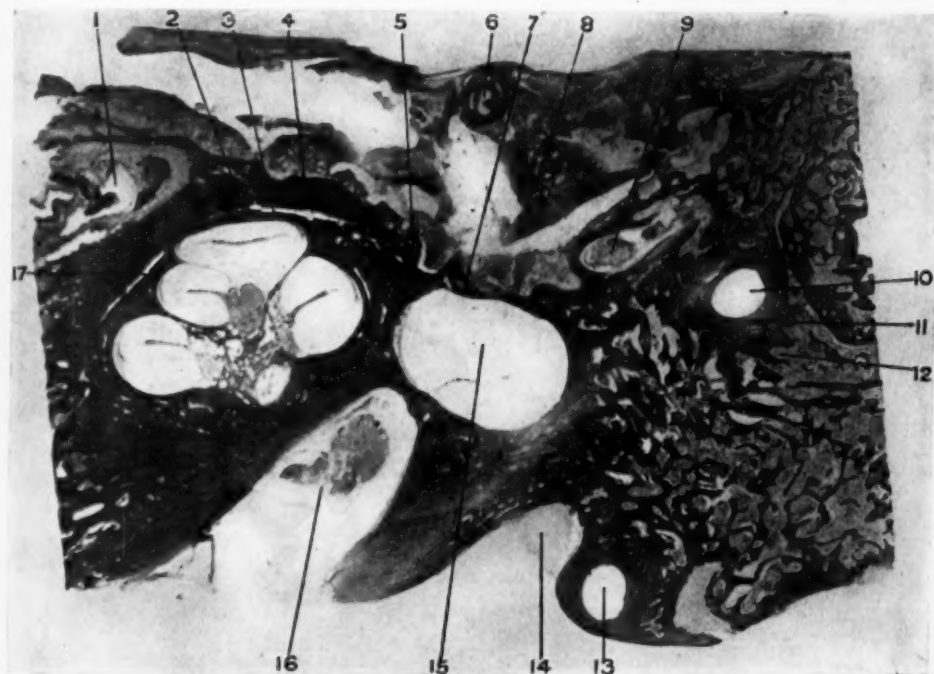


FIG. 11.

Horizontal section through right ear of new-born child, showing lymph (3) space between cartilage bone (2) and lamellar bone (4) in the capsule of the cochlea. 1, carotid canal with artery; 2, cartilage bone of cochlear capsule; 3, lymph space; 4, lamellar bone formed from deep layer of tympanic mucosa; 5, anterior margin of oval window; 6, malleus; 7, footplate of stapes; 8, incus; 9, facial nerve; 10, external semicircular canal; 11, cartilage bone surrounding canal; 12, marrow; 13, smooth end of superior canal; 14, fossa subarcuata; 15, vestibule; 16, internal auditory meatus with nerves; 17, lymph space in bony capsule of cochlea. ($\times 8$.)

In order to explain how the chronic infective condition, which invades the labyrinth capsule from the region of the promontory can reach the internal auditory meatus it is necessary to recall a few facts regarding the development of the ear. About the third month of foetal life we find the otic vesicle, now differentiated into cochlea, saccule,

NORMAL AND DISEASED OSSICLES FROM CASES OF CHRONIC MIDDLE EAR SUPPURATION.
(Figs. 12 to 15 inclusive.)

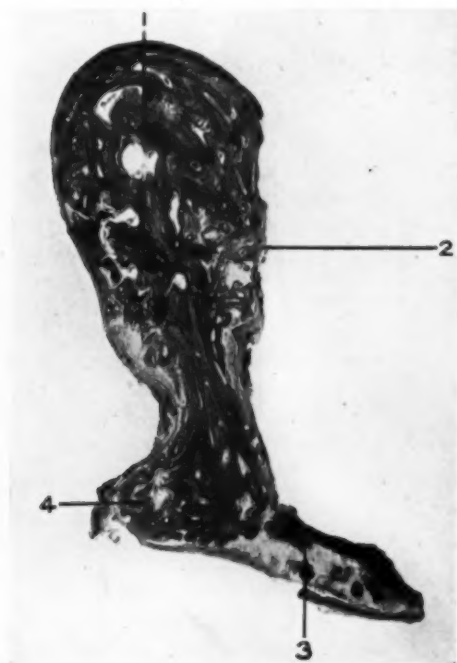


FIG. 12.

Vertical transverse section of normal malleus. 1, head of malleus; 2, articular surface; 3, handle; 4, short process. ($\times 15$.)

utricle, and canals, surrounded by a capsule of cartilage of mesoblastic origin (fig. 7). At this stage the perilymph space is still filled with foetal connective tissue. Further, the hollow bud of mucosa from the nasopharynx which lines the Eustachian tube, and is later on to line

the tympanum, aditus, antrum, and mastoid cells, has as yet only reached the anterior and lower part of the tympanic cavity. Later on, the cartilage capsule of the inner ear mentioned above becomes converted into dense, non-vascular bone (fig. 11). This bone throughout life contains numerous spaces (interglobular spaces) which hold cartilage cells even in old people.

In the child and adult the membranous labyrinth is surrounded by three distinct layers of bone (fig. 10): (1) Just outside the endosteum,

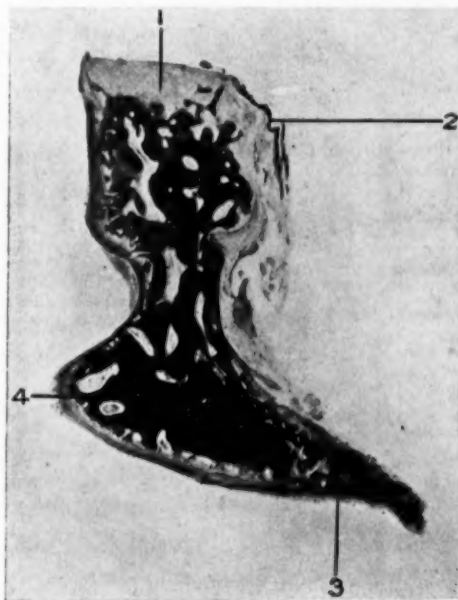


FIG. 13.

Eroded malleus from a case of chronic middle ear suppuration and cholesteatoma. 1, eroded head of malleus; 2, cholesteatoma; 3, handle of malleus; 4, short process. ($\times 15$.)

which lines the labyrinth, there is a very dense layer of compact bone formed by the outer (osteogenic) surface of the endosteum (fig. 10, No. 6); this layer is, later on, intimately fused with (2) the dense bone derived from the cartilaginous capsule of the labyrinth referred to above (fig. 10, No. 7); (3) surrounding this cartilage bone, but distinct from it, we

have the lamellar bone with its cellular, fatty, or fibrous marrow (fig. 10, No. 3). This lamellar bone, where it lies next to the middle ear, is formed by the deep layer of the tympanic mucosa and, where it lies next to the cavity of the skull, by the osteogenic layer of the dura mater (fig. 11).

Although I have not noticed it in the communications of other observers I have found that, in the fœtus, there is a well-marked space—probably a lymph space—between (2) the cartilage bone and (3) the

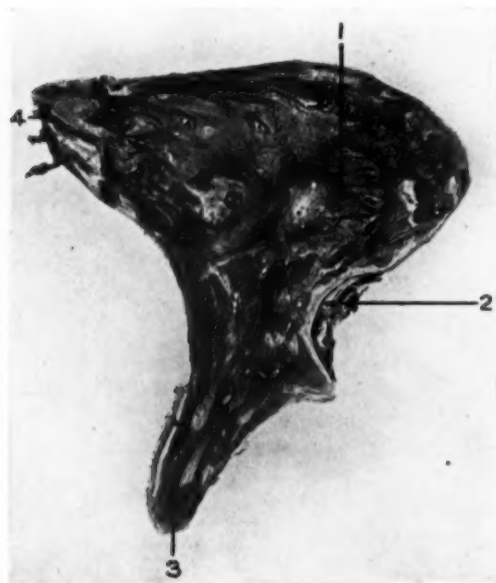


FIG. 14.

Vertical transverse section of normal incus. 1, body of incus; 2, articular surface (with blood clot); 3, long process; 4, short process. ($\times 15$.)

lamellar bone (fig. 11, Nos. 3 and 17). In young children this space is, as a rule, clearly visible though not continuous, but in later life it is not so well seen, though traces of it may still be found, especially in the capsule of the cochlea. In any case a "potential" space is always present, just as between two pieces of wood which have been glued together.

It is my belief that a chronic infective process, such as a pyogenic osteomyelitis, a syphilitic or tubercular osteomyelitis, or the condition known as otitis vasculosa, may invade the mucoperiosteum of the inner tympanic wall, infect the marrow spaces of the lamellar bone, and finally invade the lymph space described above. When once this has been opened up the chronic infective process may spread round the cochlea between the lamellar and cartilage bone, and even progress as far as the internal auditory meatus. This theory meets the objection

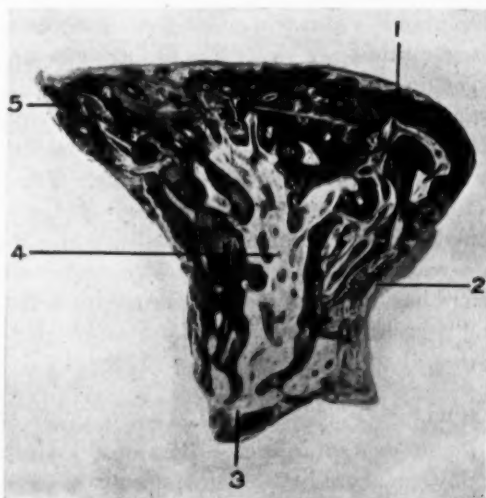


FIG. 15.

Eroded incus from a case of chronic middle ear suppuration. 1, body of incus; 2, articular surface; 3, eroded long process; 4, invasion of the marrow spaces by a chronic form of osteomyelitis analogous to that seen in the labyrinth capsule in otosclerosis and congenital syphilitic deafness; 5, short process of incus. Note large marrow space with dilated vessels extending from region of eroded long process. ($\times 15$.)

raised by Politzer—that otosclerosis cannot be secondary to an infection of the tympanic mucosa, because the otosclerotic change has been found in the region of the internal meatus, where there is no mucous membrane connected with the middle-ear spaces.

(May 19, 1916.)

Congenital Syphilitic Disease of the Ear.

By J. S. FRASER, M.B., F.R.C.S.Ed.

CONGENITAL syphilis ranks after cerebrospinal meningitis and middle-ear suppuration as the most frequent cause of acquired deaf-mutism. There is reason also to believe that many cases of so-called "congenital" deaf-mutism are really due to intra-uterine syphilis or to syphilitic changes in the ear occurring before the child has learned to talk. Statistics as to the frequency of deafness in children suffering from congenital syphilis vary very greatly. Some authorities¹ put it as high as 60 per cent., others at only 33 per cent. Yearsley finds that 3·5 per cent. of children in the London County Council deaf-mute schools are suffering from congenital syphilis, while Siebenmann, of Basle, gives a percentage of 5·6, and Beck² obtained a figure of 8·1. E. Urbantschitsch³ has examined the Wassermann reaction in 125 deaf-mutes and found it positive in thirty-three.

PATHOLOGY.

(1) *In the Fœtus and in Infants.*—Baratoux, Panse, Mayer, Asai, Hofer, Ranke and Gruenberg have microscopically examined the middle and inner ears of syphilitic fœtuses and infants. The changes found by these observers may be summarized as follows: (a) *Otitis media* is of common occurrence not only in infants but also in children born prematurely. The same may be said, however, of non-syphilitic fœtuses and infants. In syphilitic cases the infective process has been found to involve the labyrinth by rupture of the annular ligament and consequent invasion of the vestibule through the oval window. (b) *Delayed ossification* of the labyrinth capsule, with abnormal marrow spaces. (c) *Intra-uterine meningitis* in the region of the internal meatus and neuritis (small cell infiltration) of the eighth nerve. (d) Baratoux, in

¹ Carpenter, *Laryngoscope*, September, 1912.² *Münch. med. Wochenschr.*, 1910, p. 2069.³ *Monatsschr. f. Ohrenheilk.*, 1910, xlv, pp. 776, 777.

addition to purulent otitis media and interna, has noted *changes in the vessels*. (e) In numerous cases *hæmorrhages* have been observed in the middle and inner ear, but such findings must be discounted, as they are probably due to suffocation. Panse, however, holds that the hæmorrhage is of syphilitic origin. (f) Changes in Corti's organ and in other parts of the membranous labyrinth and nerve apparatus, especially the spiral ganglion, have occasionally been noted, but may be explained by post-mortem changes—at least in some cases. Gruenberg¹ has demonstrated spirochætes in microscopic sections from the ear of a seven months' fœtus, especially in the cochlear and vestibular nerves, the facial nerve, and in the tympanic and carotid nerve plexuses. Spirochætes were also found in the vessels of the middle ear and in the marrow spaces of the ossicles. There were, however, no spirochætes in the hollow spaces of the labyrinth or in the nerve endings in the inner ear. Alexander² states that the most severe forms of ear syphilis occur in intra-uterine life. The new-born infants show all the signs of congenital deafness, and the static labyrinth is not excitable.

(2) *In Young Children*.—McBride³ states that, in children suffering from congenital syphilis, what appears to be a case of simple Eustachian catarrh runs an unfavourable course. Treatment does no good, and the drumheads remain thickened and indrawn, while the deafness is severe and persists. McBride further says that in children hereditary syphilis often gives rise to a combination of middle-ear catarrh and labyrinthine deafness. Alexander holds that catarrhal otitis media is common in syphilitic children, and appears to be a syphilitic manifestation. There is only slight or no improvement after the absorption of the exudate. The middle-ear symptoms are thus replaced by those of an inner-ear lesion. *These catarrhal affections develop into conditions which clinically correspond to otosclerosis*. According to Cheatle⁴ Eustachian obstruction occurs in the "snuffling" stage of congenital syphilis. Cheatle holds that syphilis alone will not cause suppurative otitis media; pyogenic organisms are necessary. Madden (quoted by Cheatle) has recorded a case of gumma of the middle ear in a child. At the autopsy it was found that the petrous pyramid, dura mater, parietal and occipital bones were involved. From what has been said it would appear that the

¹ *Zeitschr. f. Ohrenheilk.*, 1910, lx, p. 260.

² "Ohrenheilkunde des Kindesalters," 1912.

³ "Diseases of the Throat, Nose, and Ear," 3rd ed., 1900, pp. 490, 663, 693.

⁴ *Proc. Roy. Soc. Med.*, 1911, iv (Otol. Sect.), p. 2.

basis of this variety of deafness in congenital syphilis consists of a (syphilitic ?) otitis media going on to labyrinthine involvement.

(3) *Late Type of Congenital Syphilis in which the Deafness occurs (gradually or suddenly) between the Seventh and Thirteenth Years as a rule.*—Interstitial keratitis is almost invariably present, while

CASE OF CONGENITAL SYPHILITIC DISEASE OF THE EAR.
(Figs. 16 to 25 inclusive.)

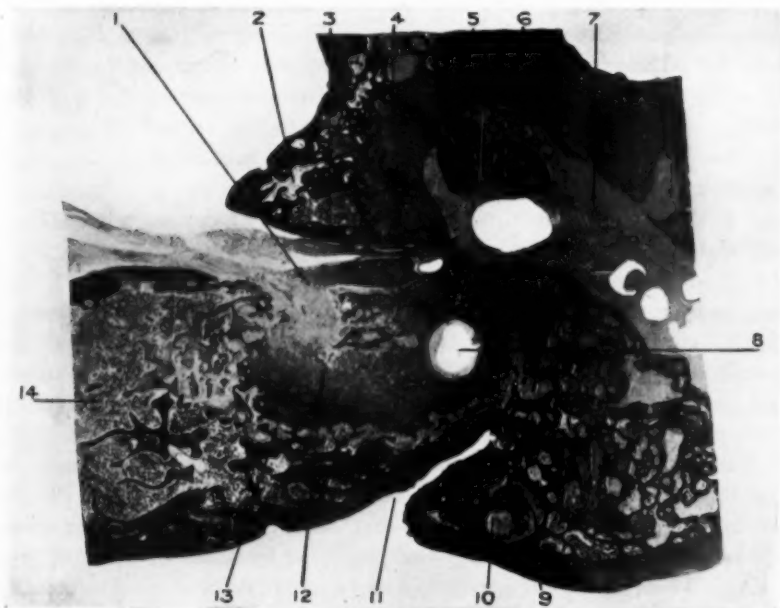


FIG. 16.

Congenital syphilitic disease of the ear. Horizontal section through left ear (No. 102). 1, geniculate bend of facial nerve; 2, normal fatty marrow; 3, normal cellular marrow; 4, osteoclastic marrow; 5, head of malleus ankylosed to attic wall—note erosions; 6, body of incus, both ossicles show wide marrow spaces; 7, greatly thickened submucosa of attic; 8, ampullary end of superior canal; 9, osteoclastic marrow eroding bony wall of canal; 10, smooth end of superior canal filled with granulation tissue; 11, opening of fossa subarcuata; 12, upper part of bony cochlear capsule; 13, osteoclastic marrow eroding cochlear capsule; 14, normal cellular marrow in anterior part of petrous pyramid. ($\times 5$)

"Hutchinson" teeth are found in 50 per cent. of cases. Various views have been expressed as to the pathology of this variety of congenital syphilitic deafness. So far microscopic examination of such cases has

been entirely lacking as far as the writer has been able to ascertain. The classical case of Walker Downie¹ is the only one that has been even macroscopically investigated. For this reason the case reported below appears to be of special interest to otologists. The views put forward by various authorities may be summarized as follows: (a) The deafness is probably due to a *neuro-labyrinthitis spreading from the meninges* (Siebenmann and Mayer). Mayer holds that the late form of congenital syphilitic deafness is due to a recurrence or an exacerbation of the condition which he and others have found in the internal auditory

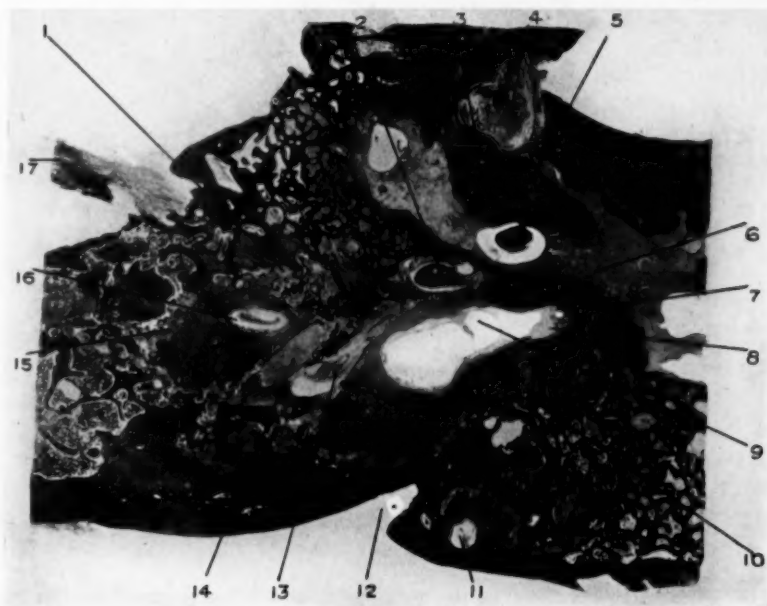


FIG. 17.

Congenital syphilitic disease of the ear. Horizontal section through left ear (No. 150). 1, osteoclastic marrow around cochlear capsule; 2, facial nerve; 3, head of malleus; 4, external meatus with desquamated epithelium and necrosed bone; 5, body of incus with enlarged marrow spaces; 6, thickened submucosa of aditus; 7, osteoclastic marrow eroding bony wall of external (horizontal) canal; 8, perilymph space of external canal filled with granulation tissue; 9, ampullary end of external canal with crista and cupula; 10, osteoclastic marrow eroding wall of canal; 11, smooth end of superior canal partly filled by granulation tissue; 12, opening of fossa subarcuata; 13, vestibular nerve; 14, facial nerve; 15, erosion of cochlea capsule by abnormal marrow; 16, basal coil of cochlea; 17, great superficial petrosal nerve. ($\times 5$.)

¹ *Archives of Otol.*, 1896, xxv, No. 1.

meatus of still-born syphilitic infants (*vide supra* [1]). The localized meningitis or neuritis is supposed to extend to the nerve structures of the inner ear. It is claimed in support of this view that Kretschner and Tobler have found lymphocytosis in the cerebrospinal fluid in older cases of congenital syphilis. (b) *Vascular changes* (endarteritis) are held by Baratoux to explain the late form of congenital syphilitic deafness. (c) *Otitis media followed by paralabyrinthitis or invasion of the*

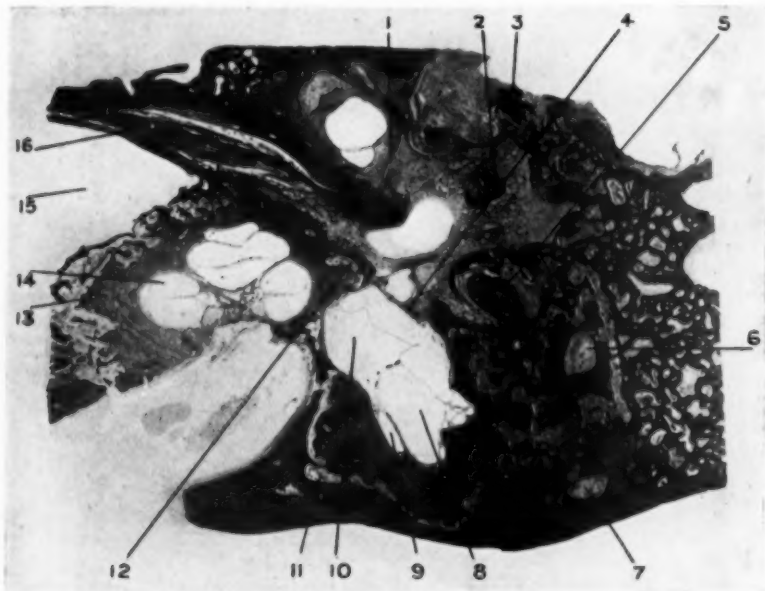


FIG. 18.

Congenital syphilitic disease of the ear. Horizontal section through left ear (No. 282). 1, tendon of tensor tympani; 2, long process of incus; 3, necrosed bone being extruded into external meatus; 4, footplate of stapes; 5, facial nerve; 6, external canal, the perilymph space is filled with granulation tissue; 7, posterior canal, also filled with granulation tissue; 8, utricle; 9, ductus endolymphaticus; 10, dilated saccule; 11, osteoclastic marrow, which, higher up, erodes the internal meatus; 12, osteoclastic marrow in bone of promontory in anterior margin of oval window; 13, bony capsule of cochlea; 14, dilated scala media of basal coil of cochlea; 15, carotid canal; 16, tensor tympani muscle. Note the erosion of the bony capsule of the cochlea by the osteoclastic marrow. ($\times 5$)

labyrinth. Almost all authorities who have written on the subject of the "late" form of congenital syphilitic deafness agree that the tympanic membranes are seldom normal. This points to a past attack

(or attacks) of otitis media. Out of thirty-three cases examined by myself the drumheads were normal in only four instances. Politzer states that congenital syphilitic disease of the labyrinth is often associated with middle-ear catarrh or suppuration. I hold that, at least in some cases, the late form of deafness is due to syphilitic otitis media—possibly with mixed infection—which (i) invades the bone of the labyrinth capsule, giving rise to a chronic form of osteomyelitis which slowly invades the labyrinth (gradual onset of deafness), or (ii) breaks through rapidly into the hollow spaces of the inner ear, causing

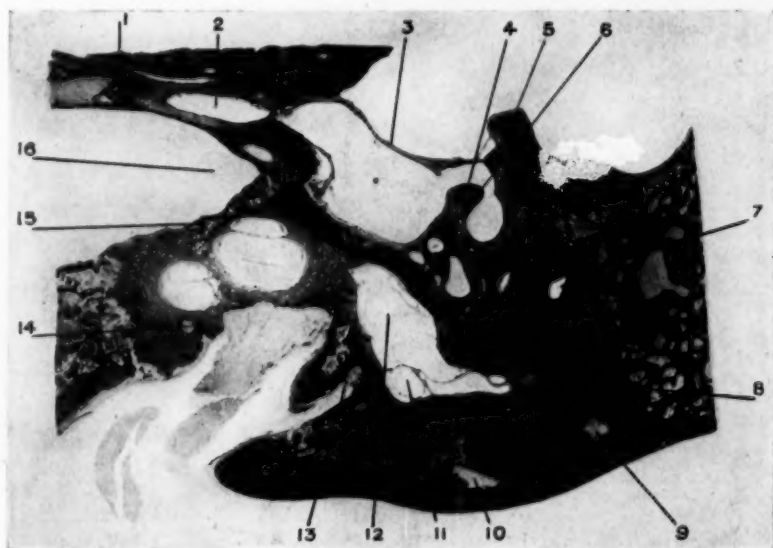


FIG. 19.

Congenital syphilitic disease of the ear. Horizontal section through left ear (No. 342). 1, junction of cartilaginous and bony tube; 2, lumen of Eustachian tube; 3, tympanic membrane; 4, long process of incus; 5, chorda tympani nerve; 6, inco-stapedial joint; 7, facial nerve; 8, perilymph space of external canal (smooth end) filled with granulation tissue; 9, posterior canal with perilymph space obliterated; 10, upper end of opening for saccus endolymphaticus, the dura has been removed; 11, lower part of utricle which joins ampulla of posterior canal; 12, greatly dilated saccule; 13, nerve to crista of posterior canal; 14, area of otitis vasculosa (chronic osteomyelitis) between basal coil of cochlea and internal meatus; 15, otitis vasculosa in anterior part of cochlear capsule between periosteal and lamellar bone; 16, carotid canal. [Between the dilated portion of the endolymphatic duct (fig. 18, No 9) and the saccus endolymphaticus (fig. 19, No. 10) the endolymph duct appears to have been occluded by the inflammatory changes in the marrow; hence the dilatation of the membranous labyrinth.] ($\times 5$.)

labyrinthitis (apoplectiform onset of deafness). In support of this view of the pathology I put forward the following case, for permission to record which I am indebted to Dr. Logan Turner:—

D. W., male, aged 20, had measles at the age of 4, followed by otorrhœa. The father died of aneurysm at the age of 35; the mother states that she had had four stillborn children before the birth of the patient; afterwards she had three more still-born children. At the age of 9 the patient had interstitial keratitis, and six months later *syphilitic ulceration of the pharynx*, followed by

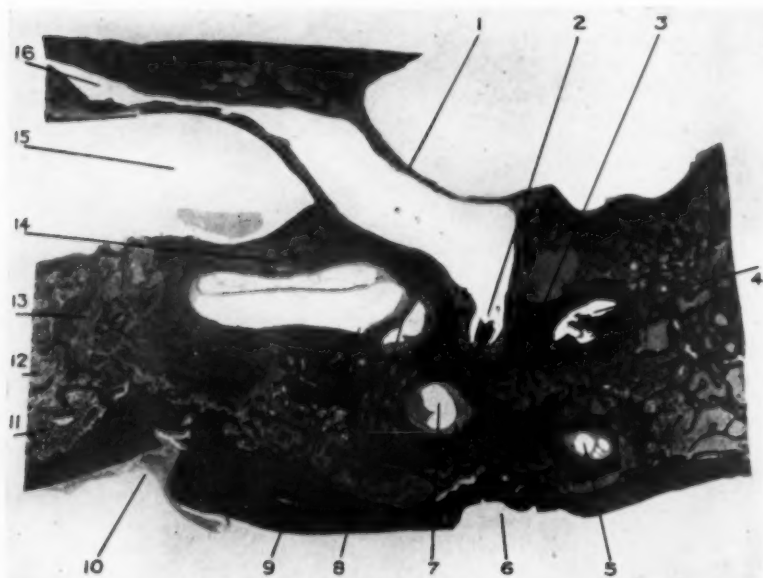


FIG. 20.

Congenital syphilitic disease of the ear. Horizontal section through left ear (No. 428). 1, Tympanic membrane; 2, sinus tympani; 3, stapedius muscle; 4, facial canal (nerve displaced—artefact); 5, smooth end of posterior canal partially filled up; 6, depression for saccus endolymphaticus; 7, ampullary end of posterior canal; 8, membrane of round window; 9, cochlear opening of perilymphatic aqueduct; 10, cranial end of perilymphatic aqueduct; 11, normal cellular marrow; 12, normal fatty marrow; 13, osteoclastic marrow; 14, area of otitis vasculosa in cochlear capsule (note the deeply staining bone in the wall); 15, carotid canal; 16, Eustachian tube. ($\times 5$.)

contraction. At this period also the boy became markedly deaf, and within a year the deafness was complete. At the age of 10 enlarged glands (syphilitic or tubercular?) were removed from the neck. The patient's speech became

very indistinct, but his mother could understand it. For some time before his admission to the Royal Infirmary, Edinburgh, the patient had been very drowsy. Examination on admission showed that he was emaciated. The incisor teeth were notched and the corneæ very cloudy. Syphilitic scars were noted round the knees. Wassermann reaction negative. The patient's breathing was so noisy as to keep other patients awake at night. Only fluid food could be swallowed on account of the stenosis of the hypopharynx. The naso-

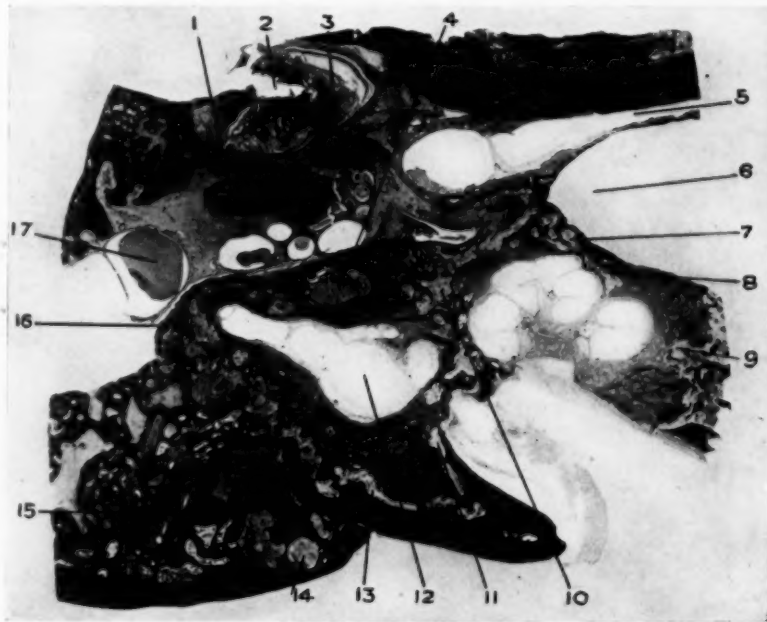


FIG. 21.

Congenital syphilitic disease of the ear. Horizontal section through right ear (No. 152). 1, incus; 2, external meatus containing desquamated epithelium and bits of necrosed bone; 3, malleus in region of short process; 4, facial nerve; 5, Eustachian tube; 6, carotid canal; 7, 8 and 9, areas of otitis vasculosa (chronic osteomyelitis) in capsule of cochlea; 10, otitis vasculosa in cochlear capsule just above and in front of oval window; 11, otitis vasculosa near internal auditory meatus; 12, utricle; 13, osteoclastic marrow (otitis vasculosa); 14, posterior canal obliterated by granulation tissue; 15, dilated vessels of fossa subarcuata, which here take a knee-shaped bend; 16, otitis vasculosa eroding bony wall of external canal; 17, cavity of aditus containing mucoid material. ($\times 5$)

pharynx was entirely shut off from the mouth and oropharynx by scar tissue. Suspension laryngoscopy was carried out and revealed marked cicatricial narrowing of the upper aperture of the larynx—the epiglottis being adherent

to the posterior wall of the pharynx. The patient died suddenly in the afternoon following this examination. The autopsy showed syphilitic changes in the skull-cap, brain membranes, lungs, heart, aorta and liver. The brain was markedly oedematous (not yet microscopically examined).

As an example of the late form of congenital syphilitic disease of the ear the case is not free from objection—e.g., (1) the Wassermann reaction was negative (this is frequently met with in cases of congenital syphilis which reach the age of 20); (2) the history and presence of suppurative otitis media (to my mind this is not an objection, but it may be to other otologists who hold a

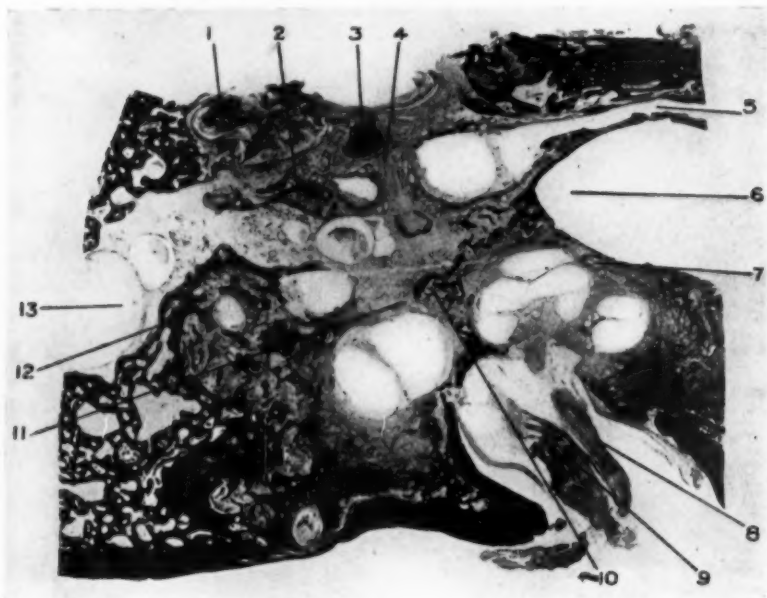


FIG. 22.

Congenital syphilitic disease of the ear. Horizontal section through right ear (No. 185). Stained with Kulschitsky hematoxylin. 1, Necrosed bone from posterior superior wall of bony meatus surrounded by desquamated epithelium; 2, incus (note large marrow spaces and erosion of the bone); 3, malleus; 4, tendon of tensor tympani; 5, Eustachian tube; 6, carotid canal; 7, area of otitis vasculosa in cochlear capsule; 8, cochlear nerve; 9, vestibular nerve; 10, area of otitis vasculosa in anterior margin of oval window; 11, footplate of stapes in oval window; 12, osteoclastic marrow reaches the endosteum of the horizontal canal; 13, mastoid antrum. Note that the nerves (Nos. 9 and 10) are apparently normal. ($\times 5$.)

different view of the pathology of "late" congenital syphilitic deafness); (3) history of enlarged glands removed from the neck (I am endeavouring to find out the details of this operation).

Microscopical examination of the middle and inner ears on both sides showed much the same conditions: (1) Purulent otitis media, with great thickening of the submucous tissues; (2) ankylosis of the head of the malleus to the outer attic wall; (3) invasion of the marrow spaces surrounding the bony capsule of the labyrinth by a chronic form of osteomyelitis, with numerous giant cells but *no caseation*; in many places deeply staining bone (hæmatoxylin) was observed in the walls of the enlarged marrow spaces; (4) marked erosion of the bony labyrinth capsule by the osteoclastic marrow; (5) invasion of the

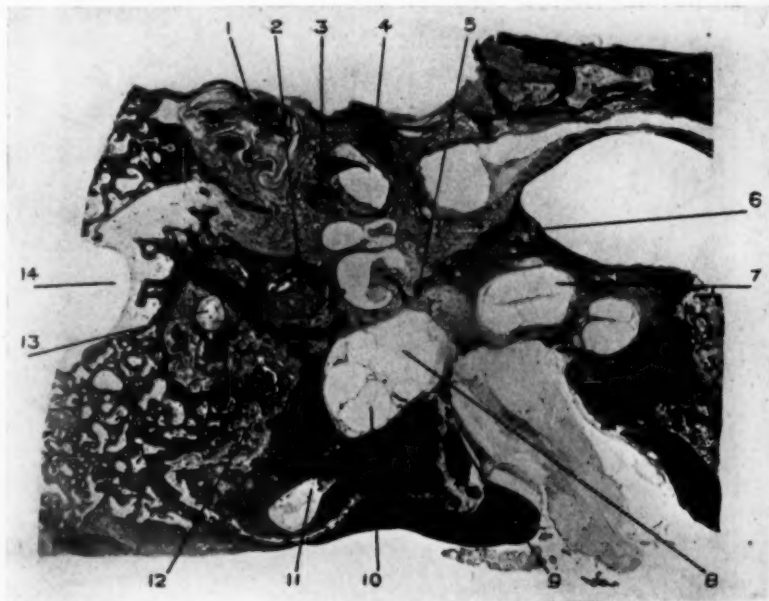


FIG. 23.

Congenital syphilitic disease of the ear. Horizontal section through right ear (No. 206). 1, Pocket in posterior wall of bony external meatus containing necrosed bone and desquamated epithelium; 2, facial nerve; 3, long process of incus; 4, malleus; 5, anterior margin of oval window; 6, area of otitis vasculosa between apex of cochlea and carotid canal; 7, dilated cochlear canal of middle coil; 8, greatly dilated sacculus; 9, marrow changes reach the internal meatus; 10, utriculus; 11, junction of smooth end of posterior canal with crus commune partly filled with new connective tissue; 12, osteoclastic marrow; 13, external canal: endolymph space patent, but perilymph space filled up: the canal is surrounded by osteoclastic marrow which reaches the endosteum; 14, mastoid antrum. ($\times 5$.)

semicircular canals by erosion of their bony walls and formation of granulation tissue in the perilymph spaces in such a way as to cause occlusion; (6) marked dilatation of other parts of the membranous labyrinth—utricle, saccule and

membranous cochlea; (7) on the right side the cochlear opening of the perilymphatic aqueduct was filled up by connective tissue; (8) the changes in the nerve endings were slight—e.g., in the cristæ of the canals, neuro-epithelium of the utricle and saccule and in Corti's organ; (9) the two divisions of the eighth nerve, along with the facial nerve, were normal (see figs. 16 to 25).

My explanation of the appearances described above is that, in the syphilitic patient, the otitis media invaded the bone of the inner wall of the middle ear, especially in the region of the external semicircular canal, and set up a chronic form of inflammation in the marrow spaces. Later on this attacked the dense

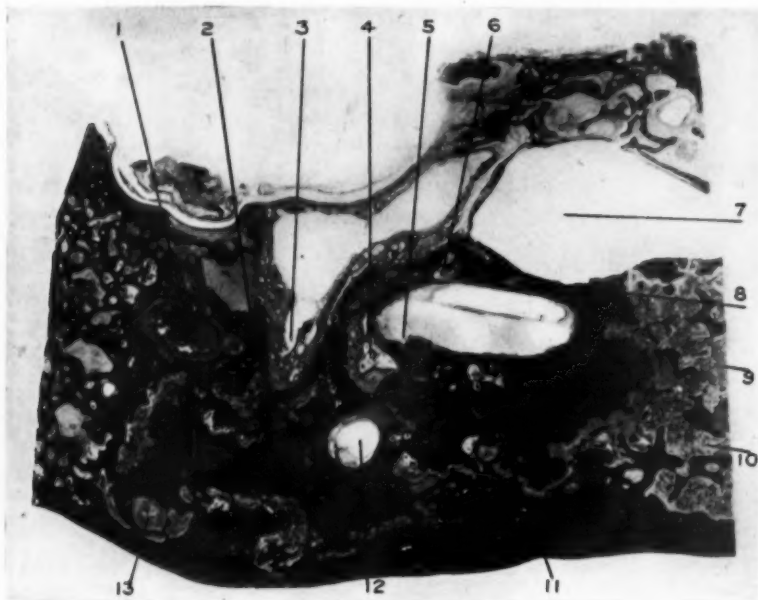


FIG. 24.

Congenital syphilitic disease of the ear. Horizontal section through right ear (No. 274). 1, Facial nerve; 2, stapedius muscle; 3, sinus tympani; 4, niche of round window; 5, cochlear opening of perilymphatic aqueduct filled up with delicate connective tissue which is only faintly seen; 6 and 8, area of otitis vasculosa in cochlear capsule; 7, carotid canal; 9, cellular marrow (normal); 10, fatty marrow (normal); 11, area of otitis vasculosa behind basal coil; 12, ampullary end of posterior canal; 13, smooth end of posterior canal obliterated by connective tissue and surrounded by osteoclastic marrow. ($\times 5$)

cartilage bone of the labyrinth capsule and eroded it, giving rise to changes in the hollow spaces of the labyrinth and obliterating in parts the external or horizontal canal. The dilatation of the membranous labyrinth is harder to

explain, but it may have been due to obstruction of the ductus endolymphaticus in its passage from the vestibule to the saccus. As yet this point has not been definitely settled.

Hennebert¹ was the first to call attention to the presence of "compression" nystagmus in some cases of congenital syphilitic disease of the ear. Increase of the air-pressure in the external meatus by means of a valveless Politzer bag causes a slow movement of the eyes to the same side, while aspiration brings about a slow movement to the opposite side. Hennebert finds that in these cases the rotation reaction is always absent, while the caloric reaction is reduced. Buys and Alexander

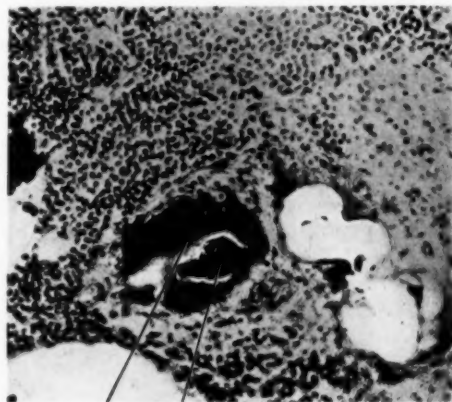


FIG. 25.

Congenital syphilitic disease of the ear. Section through osteoclastic marrow showing large osteoclast with many nuclei; the giant cell contains a bit of necrosed bone. ($\times 300$.)

have also reported cases. Alexander thinks that Hennebert's sign is due to a change in the nerve endings, while Bárány holds that it is due to excessive mobility of the stapes. I believe that my findings (*vide supra*) in congenital syphilitic disease of the ear point to another explanation—namely, that the bony wall of the external canal is eroded by inflammatory changes in the marrow and that the hollow space of the canal is partly filled up by syphilitic granulation tissue, so that the rotation reaction cannot be produced. The vertical canals may also be

¹ Hennebert, *La Presse otolaryngologique Belge*, 1909, No. 5.

affected, so that the caloric reaction is feeble or absent. The more powerful pneumatic test is, however, able to bring about an abnormal (reversed) fistula symptom.

Walker Downie's classical case appears to me to support the view held by myself as to the pathology of congenital syphilitic deafness—i.e., that the condition is due to labyrinthitis (paralabyrinthitis, empyema of the labyrinth, or panlabyrinthitis) following otitis media in a syphilitic subject. Downie's patient became deaf at the age of 11, the deafness being accompanied by *severe pain in both ears*, though there was never any discharge. (We all, however, recognize that many cases of otitis media do not suppurate or discharge.) The deafness became complete in six months. Otoscopy showed the drumheads to be indrawn and opaque. The patient died from meningo-encephalitis resulting from a fungating gumma of the right parietal bone. Macroscopic examination of the ears showed that the seventh and eighth nerves were healthy. The middle ears were normal. (We know that an otitis media may pass off and leave little or no trace.) The semicircular canals were obliterated and the vestibule largely filled up by new bone formation. The modiolus of the cochlea was thickened. These findings seem to correspond with a stage of sclerosis and ossification following an attack of labyrinthitis.

(May 19, 1916.)

Chronic Osteomyelitis of the Labyrinth Capsule (Paralabyrinthitis) in Suppurative Otitis Media.

By J. S. FRASER, M.B., F.R.C.S.Ed.

IN the microscopic examination of the middle and inner ears from nearly 100 cases of suppurative otitis media, I have come across numerous instances in which the infective process has invaded the labyrinth capsule. Several of these occurred in cases of virulent acute suppurative otitis media (streptococcal infection), but here the process was a very acute one—the bone melting away, as it were, under the action of the toxins and organisms. In chronic middle-ear suppuration, on the other hand, conditions resembling those found in otosclerosis and in the syphilitic case just reported have been noted on several occasions. The following case is an example:—

C. G., female, aged 6, had discharge from the left ear following an attack of measles at the age of 3. On December 25, 1911, she had severe pain in the left ear followed by vomiting, dizziness and headache; these symptoms gradually improved, and by January 8, 1912, she was fairly cheerful. When seen for the first time on January 13 the left external meatus contained pus and granulations; no mastoid tenderness. The tonsils were enlarged and a mass of adenoids was present. January 15, 1912: The adenoids and tonsils were removed. Unfortunately the granulations in the left ear were also curetted at the same time—a procedure which is now strictly avoided, unless the labyrinthine function has been tested and found normal. January 17, 1912: Patient cyanosed and vomiting; temperature 103.2° F., pulse 160. January 18, 1912: Vomiting continues; complete deafness in left ear; giddiness present. No rigidity of neck; Kernig's sign absent; rotatory and horizontal nystagmus to right (sound side). Pulse very feeble. Tongue dry, brown and furred; breath foul. Diarrhoea with mucous stools. Lumbar puncture: Cerebrospinal fluid under increased tension, but clear. Films show several polymorphs and some Gram-positive streptococci and Gram-negative bacilli. Operation considered hopeless. January 19, 1912: Temperature 97° F. Heart-beats (counted with stethoscope) 140; vomiting continues; restlessness; diarrhoea continues. Anti-streptococcus serum was given, also stimulants; digitalin and strychnine were injected; facial paralysis noted on left side. Death.

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Post-mortem: Cloudy swelling of kidneys, heart, liver; spleen enlarged and pale; petechial hæmorrhages in lungs; evidences of enteritis; mesenteric glands showed recent acute inflammation in addition to chronic enlargement. No obvious meningitis. The appearances were those of acute septicæmia.

The case appears to have been one of septicæmia and malignant serous meningitis following the removal of tonsils and adenoids and curettage of ear granulations in a patient suffering from latent labyrinth suppuration.

EXAMINATION OF LEFT EAR (*see* figs. 26 to 28).

On naked-eye examination the endolymphatic sac appears healthy. There is a polypus in the external meatus, and the anterior wall of the meatus is perforated. The sigmoid sinus is healthy and also the jugular bulb.

Microscopic Examination.

Middle ear: The tube is lined by columnar epithelium; the tympanum, aditus and antrum by cholesteatoma. There is caries of the bone beneath the cholesteatoma. On the inner wall of the attic there is a very vascular polypus which springs from the bone just external to the ampullated end of the superior canal. The root of the polypus extends back across the floor of the aditus. The malleus and incus have been apparently displaced backwards (curettage). The head of the stapes is absent, and the footplate appears to be necrotic and to be displaced upwards towards the tympanic cavity. There is a large perforation of the tympanic membrane. Cyst formation is seen in the sub-mucosa of the antrum. The annular ligament and the secondary tympanic membrane are swollen and infiltrated.

Labyrinth capsule: There is extensive replacement of the cartilage bone of the labyrinth capsule by granulation tissue. The inflammatory process appears to have invaded the labyrinth capsule from the inner wall of the attic. The disease has spread between the endosteal bone and the lamellar bone over extensive areas, and the endosteal bone which remains, lining the vestibule and canals, is necrotic. The facial canal and nerve are apparently healthy. There are numerous osteoclasts in the granulation tissue, which lies next to the necrotic endosteal bone in the vestibule and canals. There is an area of softening in the promontory in front of the oval window, where the inflammatory process is invading the bone from the subcutaneous tissue (fig. 27, No. 7).

Cochlea: The intravestibular part of the cochlea is filled with fibrous tissue, and the basal coil shows delicate connective tissue and a little new bone in the scala tympani and vestibuli; here there is great dilatation of the cochlear duct (fig. 28). The scala media contains hyaline substance, and a trace of Corti's organ can be seen in the middle coil, elsewhere Corti's organ is absent. The capsule of the cochlea shows several areas of *ostitis vasculosa*, especially

in the region of the anterior margin of the oval window. The ductus perilymphaticus is filled with fibrous tissue at its cochlear end, but is healthy at the cranial end.

Vestibule: This cavity contains much pus. There is a specially thick layer of pus and granulation tissue just internal to the oval window. Pus is also seen at the junction of the superior canal with the utricle. The capsule of the

CASE OF CHRONIC SUPPURATIVE OTITIS MEDIA AND CHOLESTEATOMA WITH PANLABYRINTHITIS (INFLAMMATION OF LABYRINTH CAPSULE AND OF HOLLOW SPACES OF LABYRINTH). (Figs. 26 to 28 inclusive.)

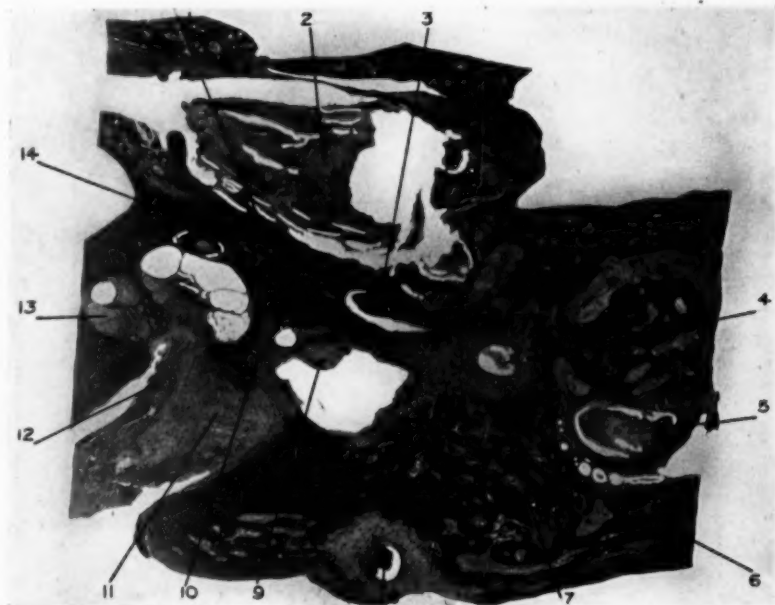


FIG. 26.

Chronic middle-ear suppuration with cholesteatoma: paralabyrinthitis and empyema of labyrinth. Horizontal section through left ear (No. 200). 1, Area of otitis vasculosa (chronic osteomyelitis) due to invasion of bone of promontory from deep layer of submucosa; 2, cholesteatoma in tympanum; 3, facial nerve; 4, horizontal canal containing pus; the endosteal bone around it is necrotic and the cartilage bone is replaced by granulation tissue; 5, cholesteatoma in antrum; 6 and 7, marrow spaces containing granulation tissue; 8, pus in smooth end of superior canal (cf. 4); 9, pus in vestibule; 10, area of otitis vasculosa; 11, vestibular nerve in internal meatus embedded in granulation tissue; this tissue may have compressed the posterior branch of the internal auditory artery; 12, cochlear nerve; 13, basal coil of cochlea; the scala tympani and the scala vestibuli are filled by connective tissue; the cochlear canal is dilated; 14, lymph space in capsule of cochlea. ($\times 5$.)

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vestibule looks as if it had been dissolved by toxic products and replaced by granulation tissue. The endosteal bone is necrotic on the inner wall of the vestibule, and also around the external canal; outside this the interglobular bone is being eaten away by giant cells. The superior and external canals show the same condition. There is erosion of the bony wall of the vestibule below the oval window and necrosis of the bony partition between the internal meatus and vestibule. The membranous structures of the vestibule cannot be recognized (figs. 27 and 28).

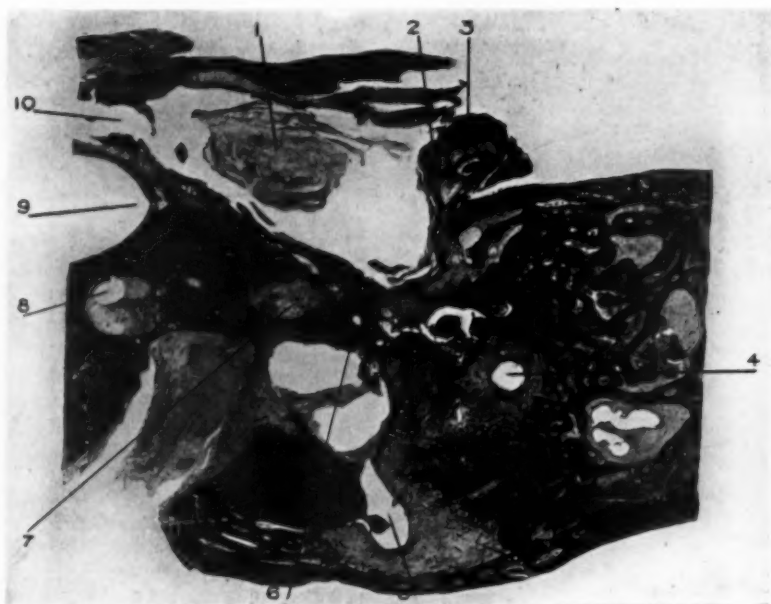


FIG. 27.

Chronic middle-ear suppuration with cholesteatoma: paralabyrinthitis and empyema of labyrinth. Horizontal section through left ear (No. 260). 1, Cholesteatoma; 2 and 3, malleus and incus displaced; 4, external canal surrounded by a ring of dead bone and a layer of granulation tissue; 5, crus commune containing pus (cf. 4); 6, footplate of stapes displaced outwards from oval window; 7, intravestibular portion of cochlea filled with granulation tissue; 8, dilated cochlear canal; 9, carotid canal; 10, Eustachian tube; the outer wall (upper in the photomicrograph) is lined by cholesteatoma. ($\times 5$.)

The canals show much the same changes as those in the vestibule—i.e., eating away of cartilage bone by granulation tissue containing giant cells and necrosis of endosteal bone. The perilymph spaces of the canals

contain pus. The outer wall of the external canal is necrosed, but there are traces of the membranous canal present. The granulation tissue around the posterior canal is in contact with the dura of the posterior cranial fossa. The posterior canal is full of pus and the bony walls of the canal show the changes described above. Near the ampullary end the bony wall is almost normal.

Internal meatus: Pus and granulation tissue are seen along the branches of the vestibular nerve to the superior and external canals. The vestibular

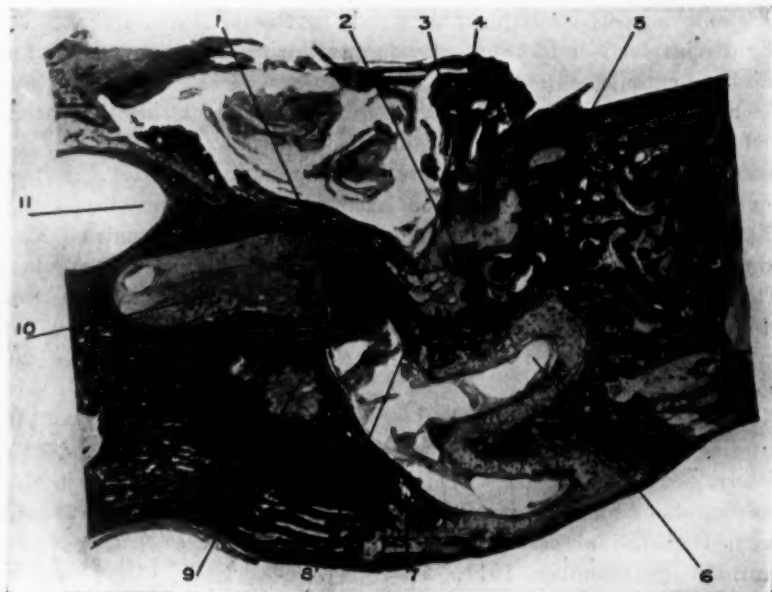


FIG. 28.

Chronic middle-ear suppuration with cholesteatoma: paralabyrinthitis and empyema of labyrinth. Horizontal section through left ear (No. 310). 1, Basal coil of cochlea in region of round window filled with connective tissue; 2, stapedius; 3 and 4, displaced ossicles; 5, facial nerve; 6, opening of external canal (smooth end) into vestibule; 7, ductus endolymphaticus; 8, area of otitis vasculosa in labyrinth capsule internal to sinus tympani; 9, large area of otitis vasculosa behind basal coil of cochlea and below internal auditory meatus; 10, new bone formation in the connective tissue filling scala tympani of basal coil; 11, carotid canal. ($\times 5$.)

nerve and ganglion are surrounded by granulation tissue, but the cochlear nerve is more normal. The bone surrounding the internal meatus is softened.

REMARKS.

The condition of the internal meatus is of considerable importance. There appears to have been abscess formation in the posterior part of the meatal fundus—a condition described by Politzer. The abscess was followed by the formation of granulation tissue, and probably by interference with the blood supply of the bony capsule of the vestibule and canals. This would favour the spread of otitis (paralabyrinthitis). The anterior part of the meatus containing the cochlear nerve was but slightly involved—a fact that may account for the healthier condition of the bony capsule of the cochlea.

From the clinical history of the case it is evident that the patient had an attack of labyrinthitis on December 25, 1911. When the patient was seen for the first time on January 13, 1912, the inner ear had passed into the stage of latent labyrinth suppuration. The changes in the labyrinth capsule also appear to have existed for a considerable period, as giant cells were present in large numbers. The paralabyrinthitis appears to have originated on the inner wall of the attic in the region of the ampullary ends of the superior and external canals, where there was marked erosion of the bone with polypus formation. The extension of the paralabyrinthitis was probably connected with the abscess formation in the fundus of the internal auditory meatus referred to above. It is not possible to determine the exact date of this abscess formation in the internal meatus, but from the appearance of the granulation tissue it would seem that the abscess formed at the time of the onset of labyrinthitis in December, 1911.

On the other hand the dislocation of the three ossicles and the recurrence of acute purulent labyrinthitis in the cavity of the vestibule and canals appear to have been due to the curettage of the tympanic cavity on January 15, 1912. "Adenoids" and tonsils were also removed at the same time. The child died from septicæmia a few days later. This seems to have been due to infection following the operation on the fauces and pharynx in such a septic case. Had the labyrinthitis alone been responsible one would have expected purulent meningitis, but not the extensive changes in the internal organs typical of acute septicæmia.

CONCLUSIONS AS TO THE PATHOLOGY OF OTOSCLEROSIS, CONGENITAL SYPHILITIC DEAFNESS, AND PARALABYRINTHITIS.

In some cases, at any rate, of otosclerosis and congenital syphilitic ear disease, as well as in many cases of pyogenic and tubercular infection of the middle ear, the chronic inflammatory process invades the bone of the labyrinth capsule and gives rise to more or less extensive paralabyrinthitis. Such a condition may in time involve the membranous labyrinth and so cause deafness. In the case of tubercular and pyogenic infections, however, it is much more common for the inflammatory process in the middle ear to penetrate the oval or round windows and so produce a rapid loss of labyrinthine function.

I wish to tender my best thanks to Mr. Richard Muir for the great care and skill he has expended on the photomicrographs illustrating this communication. I also desire to acknowledge a grant from the Carnegie Trust towards the expenses of the investigation.

DISCUSSION.

The PRESIDENT: These specimens are particularly interesting to me, especially the one in which Dr. Fraser described otosclerosis arising in the course of suppurative middle-ear disease. Clinically, some years ago, I concluded that there was more frequently an association between suppurative middle-ear disease and otosclerosis than is commonly supposed, but I have never found a case in which one could demonstrate it pathologically: and I am very grateful to Dr. Fraser for having shown what I believe to be the first case which proves the relationship between the two. Clinically, as one knows, one frequently meets cases of extreme deafness in comparatively young people associated with suppurative middle-ear disease, but the degree of the deafness is out of all proportion to the middle-ear condition. In these cases it is very apt to be assumed that the condition is one of pure middle-ear deafness, the result of suppurative middle-ear disease. When I studied the question of heredity in relation to otosclerosis, I found there was sometimes a curious relationship: I found two or three families in which there was marked otosclerosis, and in some of those families one or two individuals had suffered from suppurative middle-ear disease. In such circumstances I found that when middle-ear disease occurred in such families, it called otosclerosis into existence at a much earlier time in life than amongst other members of the family, and the deafness reached a higher degree. The middle-ear inflammation

appears to cause antedating of the otosclerosis. But we must be very careful about assuming that the otosclerosis is therefore necessarily due to infection proceeding from the middle ear: I think all it does is to excite the condition in those predisposed to it. The difficulty about the ideas of speaking of otitis media or osteitis in otosclerosis lies in the fact that we find utterly different changes in the other parts: there is an atrophy of the ceruminous glands, a diminution in the sensitiveness of the tympanic membrane, of the meatus, &c., and I think it quite probable that there are changes in the nerve-cells of the cerebral cortex or other portion of the auditory tract which may account for the tinnitus. I do not think the tinnitus is explained by the changes in the internal ear. In three out of the four cases I had, the internal ear appeared perfectly normal.

Dr. P. WATSON-WILLIAMS: It is very interesting and a great privilege to see such a splendid series of microscopical specimens, which represent enormous labour. I have long been rather attracted by the suggestion which Dr. Fraser has put forward as to the possibility of otosclerosis being, at any rate in some cases, due to toxic absorption or to some chronic inflammatory process. It is a view which I have myself taught and advocated. For the last six or seven years I have been endeavouring to see how far one could obtain evidence of such source of infection in the posterior ethmoidal cells or the sphenoidal sinuses, because I think it is as reasonable to look to these parts for the source of infection as to the ear itself. One often encounters cases of pure otosclerosis in which signs or history of inflammatory ear conditions are entirely absent. And in a certain proportion of cases careful investigation has led to my discovery of chronic or very latent infection of sinuses. It may so happen that we may have to look for other foci as possible sources of infection. But it seems that, just as in cases of canicular optic neuritis following on sphenoidal sinus infection, the infection may be very slight indeed, and sometimes it is only by means of delicate cultural methods that it can be demonstrated that there is any infection of these cavities. And we may explain the marked toxic infection where there is such a marked absence of suppuration as being due to the fact that there is no phagocytosis because of the poverty of leucocytes. Therefore instead of a suppurative process and grosser evidences of infective inflammation, there is simply a comparatively uninhibited chronic infection. The condition just mentioned may also set up otosclerosis, and yet the chronic infection may be overlooked owing to the absence of pus or other gross evidences of infective inflammatory processes.

Dr. DUNDAS GRANT: Dr. Watson-Williams has touched on a point of great practical importance—namely, that otosclerosis may depend upon an absorption of toxins. There may be many different causes of otosclerosis, which it would be impossible now to discuss. At one time I formed the impression that it occurred in association with chronic osteo-arthritis. Many cases may be of that nature. The ætiology of otosclerosis is very

various, and Habermann, in a long paper, associated it in some cases with tuberculosis, in others with syphilis. Dr. Fraser has referred to Hennebert's observation that in cases of congenital syphilis there might be the "fistula" symptom, and I would ask Dr. Fraser whether he thinks it possible that such softening of bone as he describes may be sufficiently great to permit of it yielding under any pressure which can be exerted by means of the Politzer bag in the external meatus. We are very much indebted to Dr. Fraser for his very clear demonstration.

Dr. DAN MCKENZIE: I join in thanking Dr. Fraser for a very excellent exhibition of pictures, and congratulate him upon his industry, and upon his opportunities which that industry has enabled him to utilize to the full. Turning to Dr. Fraser's theoretical allusions, I am inclined to the view that the changes which he has shown, which resemble those of the classical otosclerosis, are really changes similar to those, but the result of different causes. I take it that bony changes may show resemblance to each other and yet be due to diverse causes; and I question, therefore, whether the specimens he has shown us are sufficient to displace the original idea of classical otosclerosis being unconnected with inflammatory change. That connexion must be actually demonstrated before we can agree. I do not know whether Hennebert's observations have been confirmed. ["Yes."] It has often occurred to me that the secretory changes, to which the President has alluded, which take place in otosclerosis may be due not so much to a common origin with the otosclerotic changes in the labyrinth capsule as to the secondary effect of the abolition of function. I think secretory changes of that description do occur reflexly at times.

The PRESIDENT: One can understand them occurring reflexly, not from changes in bone, but from changes in the nervous system. I could not imagine a bone change in the labyrinthine capsule causing diminution in the secretion of wax.

(May 19, 1916.)

Case of Latent Tuberculosis of the Lateral Sinus Secondary to Chronic Suppurative Otitis Media ; Histological Specimen of the Sinus Contents.

By SYDNEY SCOTT, M.S.

A LITTLE girl, A. C., was aged 2½ years when discharge from the right ear began. At the age of 2½ years caseous cervical glands from the right side and a large mass of adenoids were removed. When aged 5½ years, a mastoid abscess appeared on the right side, and a simple mastoid operation was performed. The wound healed except for a small sinus. She was first seen in consultation six months afterwards, when the sinus was still present, together with discharge from the ear, which had never ceased. There was also a deep-seated gland behind the angle of the jaw on the same side. The child was plump but pale; her daily temperature for three weeks was between 99° F. and 100° F.; the tongue was slightly furred. She had complained a little of headache at times, but she had not vomited nor had she had fits or rigors, and though quiet would run about and play as usual in or out of doors. The tympanic membrane was obscured by pale granulations deep in the meatus. Otherwise there was no other evidence of disease in other parts of the body.

Operation: Schwartz's operation was performed, but more extensively than appeared to have been done before. The dura mater was exposed posteriorly, and here a considerable circular area (3 cm. or 4 cm. in diameter) was found covered with pale gelatinous granulations, which had a close resemblance to a cerebellar hernia. However, when the bone was freely removed and the granulations between the occipital bone and the dura mater were scraped away, it was seen that the dura mater itself was intact, and that the lateral sinus did not contain blood. The sinus was thereupon opened and found to contain an adherent fibrous mass, with loculi of pus and caseous débris. This was removed by curetting, and the sinus exposed back to the torcular before a normal condition was reached. Here free bleeding took place, and this was arrested by inserting a muscle-graft into the sinus, a short distance from

the torcular. The upper entrance to the jugular bulb, but not the bulb itself, was then exposed. There was no bleeding from this end. Pale granulations were curetted out of the antrum, but the incus, which was seen, was not disturbed, neither was the malleus nor tympanic membrane touched. The posterior wall of the bony meatus was removed, leaving the tympanic ring only, but the fibro-cartilaginous meatus was left intact. The skin incisions came well together and were sutured, only a small opening for a drainage-tube being left in the antral region. At this time it was anticipated that some further operation would probably be required to deal with the contents of the tympanum, to expose the jugular bulb, and remove the deep-seated glands in the upper part of the neck, but as the wound healed and the otorrhœa ceased, and the granulations disappeared from the meatus, nothing more has been done. It is nearly a year since the operation, and there is no sign of any return of the disease. Moreover, the cervical glands are no longer palpable.

The histological specimen (shown) demonstrates the contents of the adherent and organized clot in the lateral sinus, with many fibro-caseous tubercles and numerous giant cells.

(May 19, 1916.)

Case of Double Acute Otitis Media complicated by Ulcerative Endocarditis; Death.

By E. D. D. DAVIS, F.R.C.S.

AN unhealthy youth, aged 16, attended the out-patient department on April 7, complaining of a discharge from both ears and of recent earache. Five weeks previously he had had so-called influenza, and when seen on April 7 both ears had been discharging freely for a few days, but there was no pain and no sign of mastoid involvement, though the temperature was 101.6° F. Suitable treatment was prescribed, and, three days later, he returned with a temperature of 103° F., with tenderness, redness, and slight œdema over the lower portion of the left mastoid process. A simple mastoid operation was performed almost immediately, but only a little muco-pus was found in a large apical

mastoid cell. The mastoid antrum and lateral sinus were normal. The day after operation the temperature was normal, but it rose again on the second day to 104° F. The patient was thoroughly examined by a physician and no cause for the fever was discovered. The mastoid wound was normal.

On April 21, ten days after the operation, a deep-seated swelling was found in the left forearm in connexion with the ulna. This was punctured with a needle and some pus aspirated, which produced streptococci. The patient had no rigors and denied that he had any pain. The forearm abscess was an accidental discovery. The mastoid wound had practically healed and was normal, but pus from the wound and the blood contained streptococci on cultivation. The blood count was normal.

At this time a murmur was discovered on auscultation of the heart, and a history of rheumatic fever six years ago was obtained. The diagnosis of ulcerative endocarditis was established; embolism of the left posterior tibial artery occurred, followed by gangrene of the foot, and the patient died one calendar month after the simple mastoid operation.

It is assumed that the mastoid operation was unnecessary, but the relatives positively refused a post-mortem examination.

DISCUSSION.

The PRESIDENT: The endocarditis was probably also secondary to the streptococcus infection, and I think it is very probable that the boy had some valvular lesion before, resulting from the rheumatic fever five years ago, and that the streptococcus found a suitable nidus on the valvular lesion. Many physicians hold that ulcerative endocarditis is not a specific disease in itself, but that it may occur whenever there is a septic infection, especially in those who have a valvular lesion.

Mr. DAVIS (in reply): A physician examined this case thoroughly, but he may have overlooked the cardiac lesion. Until this boy had influenza there were no signs, and it is possible rheumatic fever would account for the weak spot in the endocardium.

(May 19, 1916.)

An Obscure Complication of Middle-ear Suppuration.

By E. D. D. DAVIS, F.R.C.S.

A BOY, aged 16, was sent to hospital by his doctor with a diagnosis of brain abscess. He had been ill for fourteen days with severe headache, occasional vomiting, and a discharge from the ear of indefinite duration. He was seen on admission by a surgeon, who decided to "wait and see." Three days later the temperature was 105.4° F., respiration 40, pulse 130. He looked ill and complained of pain in the chest. The right ear was discharging freely, the surface of the mastoid process had been blistered before admission, and as far as could be ascertained there was no definite sign of mastoid suppuration.

An examination of the chest revealed signs of consolidation of the left lower lobe of the lung, with a localized pleuritic rub; in addition there was a history of a rigor. The diagnosis of lateral sinus thrombosis was made and I operated on the mastoid at once.

At the operation the mastoid antrum was found to be deeper than the lateral sinus, and overlapped by it, and to contain pus and granulation tissue. The sinus was exposed for 1 in., but no pus was present in its groove. Then a gauze plug was inserted at the upper end of the sinus groove for compression before opening the sinus; it was seen that the sinus was not thrombosed, as it readily collapsed and deflated, so to speak. The sinus was freely incised, and, on removal of the gauze, extensive hæmorrhage occurred.

On the day after the operation the temperature fell by crisis, and has remained near the normal for more than a week, and the patient appears to be practically well. Free hæmorrhage occurred when the gauze plugs were removed forty-eight hours after operation. At the time of the operation it was felt that there was not sufficient cause in the mastoid for the serious condition of the patient, and the physical signs in the chest were possibly due to lobar pneumonia. Lumbar puncture produced normal and sterile cerebrospinal fluid.

(May 19, 1916.)

**Case of Fracture of the Base of the Skull in a Baby,
aged $1\frac{3}{4}$ Years.**

By E. D. D. DAVIS, F.R.C.S.

A LITTLE girl, aged $1\frac{3}{4}$ years, was seen on account of acute otitis media with hæmorrhage from the left ear. The child looked well, but there was a copious blood-stained discharge from a perforation in the lower half of the left drum. The temperature was normal. Three days later the child was standing on her bed and fell to the floor on to her head. The right forehead was extensively bruised, and a profuse discharge of clear cerebrospinal fluid from the right ear commenced. When she was seen four days after the fall the dressings were soaked with cerebrospinal fluid which could have been collected in a test-tube. When the child cried the fluid pumped out of the ear, and it was not possible to see the drum. The temperature had risen to 103° F. on the fourth day, and a further report of the case will be given later. Apart from the discharge of cerebrospinal fluid there were no other signs of a fracture of the skull. The left otitis media had improved considerably.

Mr. DAVIS: I heard yesterday that the child was doing well, and that the temperature was normal, but there was some rigidity of the neck; and I have been expecting all the week to hear that meningitis has developed, and that trephining would be necessary. Should meningitis occur, I think one would be justified in opening the middle and posterior fossæ for drainage.

Note by Mr. Davis.—The course of the case is now complete. The child died from meningitis sixteen days after the accident. Operation was refused.

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PATHOLOGICAL SECTION.

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Pathological Section.

President—Dr. F. W. ANDREWES, F.R.S.

(May 2, 1916.)

Notes on the Bacteriological Examination of Eleven Cases of Cerebrospinal Fever treated in St. Bartholomew's Hospital during the First Three Months of the Year 1916.

By F. W. ANDREWES (President).

THERE are still certain points about cerebrospinal fever not finally settled, and it is therefore worth while to record the facts which we have noted in a series of eleven consecutive cases at St. Bartholomew's Hospital during the recrudescence of the epidemic in the winter and spring of the year 1916. The work has been done by Dr. R. G. Canti and myself. I have to thank the various physicians of the Hospital for permission to give the data of the cases under their care. Of the eleven cases, one was a soldier and ten were civilians. Of the civil cases, three were adults, three were aged 7 to 17, one was a child aged 3, and three were babies under 1 year. I do not know how to determine whether the juvenile cases should be regarded as belonging to the epidemic disease or to the posterior basic meningitis of infants. Probably no such distinction can be drawn.

In all cases diagnosis was made by cultivation of the meningo-coccus from the cerebrospinal fluid, and, in addition to routine tests, the race of the coccus was determined by agglutination. The sera employed were furnished by the kindness of Lieutenant-Colonel Gordon from the central cerebrospinal fever laboratory at Millbank, together with a serum which I prepared from a strain isolated from one of the cases studied. In his work at Millbank last year, Gordon distinguished

four epidemic races of the coccus by the absorption of agglutinin test. Others have only been able to distinguish two races, and the question is not yet finally decided. Gordon's Types I and III are admittedly closely related and are usually clumped by the same serum; they can only be distinguished by the absorption test. His Type IV appears of very exceptional occurrence.

All our cases are referable to Gordon's Types I and II (by far the commonest), with one exception, which refused to agglutinate with any of the six sera tried. The two earliest cases in our series (onset December 26 and 27, 1915) were examples of Type I; the later cases were all Type II, with the exception noted above. It is curious that, in last year's epidemic, Type I seems to have preponderated in the earlier cases and Type II later. So far as agglutination goes, Type II does not appear quite a homogeneous group. I have had to use three sera of this type, and some strains have been clumped by one and not by the others; the absorption test, according to Gordon, shows, however, that they belong to one group. It must be noted that in the grouping of the races of such an organism as the meningococcus on serological lines, three distinct properties have to be taken into account—namely: (1) agglutinability, (2) agglutinogenic power, and (3) capacity for absorption of agglutinin. In a doubtful case all three properties should be estimated before arriving at a decision. In the present series of cases all the forms of meningococcus isolated have been shown to belong to two known epidemic strains, with one single exception, so far unplaced. This was from an infant, aged 8 months.

In all our cases the pharynx was examined for the meningococcus by means of West's swabs, the plating being carried out on legumin-trypsin-agar (trypagar) to which ascitic fluid and a little blood were added. Very different statements are current as to the frequency with which the meningococcus can be found in the pharynx in actual cases of cerebrospinal fever. Many observers in last year's epidemic failed to find it in the majority of cases examined; others have found it with some frequency. Dr. Canti and I have found it in all cases, and this without serious difficulty. In four out of eleven cases the meningococcus was the prevailing organism in the pharynx, twice in almost pure culture; in three cases it formed 5 to 10 per cent. of the colonies present, in the remainder 1 per cent. or less. In one child it was not found till the third attempt. The identity of the organism recovered from the pharynx with that obtained from the cerebrospinal fluid was established in all cases by the agglutination test. The two were always tested side

by side and found to give the same serological reactions, with, at most, slight quantitative differences. This identity of type, in every case, between the spinal and pharyngeal strains appears a point of some importance, since it indicates that the pharyngeal infection is part and parcel of the disease.

Most of the cases continued to be carriers for some weeks. Four of the cases which recovered gave negative pharyngeal swabs after twenty-eight, thirty-three, forty and fifty-one days from the onset, but in another the patient was still a carrier after seventy days.

TREATMENT AND RESULTS.

All cases were treated by lumbar puncture, frequently repeated where necessary, and by serum administered intrathecally and sometimes intravenously too. It may be remembered that in last year's epidemic serum treatment proved a disappointment. The available sera (Flexner's, Mulford's, Burroughs Wellcome's, and Lister Institute) appeared to have little influence, in most cases, on the disease. The serum which is now issued by the Lister Institute has been prepared with a large variety of strains of the meningococcus isolated during last year's epidemic and appears to be of considerable therapeutic value. At St. Bartholomew's Hospital we have been privileged to use it in all cases, and the results have been on the whole very good.

Of our eleven completed cases seven are now well and four have died. It may be said that a mortality of 36·36 per cent. is nothing to boast about, but a closer examination of the facts shows that it is really very good. Of the four fatal cases, two were infants under 1 year old—an age at which recovery from the disease is uncommon, while a third was one of very acute type, with meningococcal septicæmia, fatal on the fourth day, in a child aged 3. Of the seven cases over 4 years of age only one died—a woman aged 33; so far as her primary infection was concerned she was cured, but she developed otitis media and mastoid suppuration due to streptococcal infection, and died of streptococcal septicæmia two months from the onset of the disease. No meningococci had been present in the cerebrospinal fluid or pharynx since the third week of her illness, but at death there was matting at the base of the brain with some internal hydrocephalus.

In the cases over 4 years of age the mortality was thus only 14·3 per cent., and this contrasts very favourably with the mortality in military cases last year, which approached 50 per cent. No one who

saw the cases could have much doubt that the serum was largely responsible for this good series of results. In two adults the new serum was not given till the thirteenth and seventeenth days respectively; both were severe cases and one had been given eight doses of Mulford's serum without effect; the immediate improvement which followed a single intrathecal dose of 30 c.c. of the new serum was striking in each case. In three cases in which the serum was given on the second and third days of the disease the effect was even more remarkable, the patients being convalescent within forty-eight hours. All three were severe cases, unconscious on admission and with well-marked rashes; in one, a boy, aged 7, blood culture revealed the meningococcus in every one of six culture tubes. In two of these the serum was given intravenously as well as intrathecally—a plan which appears a good one in severe cases. In the two remaining patients who recovered, the administration of the serum produced no such marked effect, the improvement being gradual, but it is noteworthy that one of these cases was in a baby only 5 months old; in this child the disease lasted some three weeks, although serum was given on the second day and thenceforward on four more occasions.

Two accidents of some interest occurred in connexion with the administration of serum. In a man, aged 24, 100 c.c. of cerebrospinal fluid were drawn off somewhat rapidly, under an anæsthetic, and 30 c.c. of serum run in. Shortly after, while he was recovering from the anæsthetic, he had an epileptiform convulsion, perhaps due to the too rapid alteration of intracranial pressure. No ill-effects followed. In a baby, aged 8 months, admitted on the tenth day of the disease, with severe opisthotonos, repeated intrathecal doses of serum produced no improvement. As a last resort intravenous injection of serum was decided on, but as eight days had elapsed since the last intrathecal dose, anaphylactic shock was feared. Desensitization was therefore attempted by giving 1 c.c. of serum subcutaneously the night before the intravenous injection, which was run into the vein through a needle and rubber tube. Nevertheless marked shock occurred when 5 c.c. had been introduced, the child becoming blue and pulseless. As the arterial pressure fell the venous pressure rose, and blood regurgitated along the needle and rose to some height in the tube. The condition soon passed off. The child ultimately died on the forty-fourth day of the disease, with extreme internal hydrocephalus.

Blood culture was done in only three cases during life. In one, already mentioned, the meningococcus was readily obtained; this was

on the second day of the disease in a boy, aged 7, with a well-marked rash, but not otherwise a very severe case; he recovered. In the other two cases blood culture was negative on the third day of the disease. In a case in a child, aged 3, fatal in four days from onset, a single colony of the meningococcus was recovered from the blood after death though the body had been frozen two days.

SUMMARY OF CASES.

Case I.—Male, aged 44; soldier, infected at Pirbright. Onset, December 27; admitted December 31. Lumbar puncture on January 2, 6, 7, 8, and 9, but no serum till January 9, when 30 c.c. new Lister Institute serum was given intrathecally, and a similar dose on January 12. A typical moderately severe case; little improvement till first dose of serum, which was followed by great change in mental condition and fall of temperature. Temperature normal by January 12. Convalescence uninterrupted. Meningococcus from cerebrospinal fluid and pharynx proved to be of Gordon's Type I. On January 5 pharynx yielded a nearly pure culture of the meningococcus. On January 27 about 30 per cent. of the pharyngeal colonies were meningococci. On February 16 the pharynx was negative.

Case II.—Female, aged 21. Nurse at Children's Hospital, Shadwell, whence two cases of cerebrospinal meningitis had been notified on December 6 and 30 (the latter due to meningococcus of Type II). December 23: Laryngitis and supposed influenza. December 30: Onset of meningeal symptoms; admitted with a profuse rash. Lumbar puncture daily and eight doses of Mulford's serum (15 c.c.). No improvement till January 16, when she had 30 c.c. new Lister Institute serum, followed by immediate improvement and return of mental condition to normal. Sharp relapse on January 24, lasting five days. Further doses of Lister serum: 30 c.c. on January 24, 26, and 29, and 15 c.c. on February 3. This case underwent lumbar puncture on thirty occasions, always under general anaesthesia. Recovery was complete, but convalescence very protracted. On January 5 the meningococcus was abundant in the pharynx, and there were still a few colonies on March 9. Both pharyngeal and spinal strains were found to be of Gordon's Type II.

Case III.—Male, aged 11. Onset December 26, four days after leaving school for holidays. Treated at first by lumbar puncture only; no serum till nineteenth day, when he had 15 c.c. of Mulford's, which appeared to produce a good effect. Later, he had one dose of new Lister serum (15 c.c.). The case ran a very protracted course; for weeks after the temperature was normal he remained ill and helpless, with photophobia, frequent vomiting, and occasional fits of unconsciousness, with a frequent and irregular pulse. He gradually recovered during March. On January 5 the colonies from the pharynx showed

10 per cent. of meningococci; these, and those obtained from the cerebrospinal fluid, proved to be Gordon's Type I. The pharynx and cerebrospinal fluid were both free from meningococci on February 4.

Case IV.—Female, aged 33. Onset February 6; admitted February 7, with a profuse petechial rash and unconscious. Frequent lumbar puncture; 30 c.c. new Lister serum on February 8 and again on February 10. On February 14 and 16, 15 c.c. were given: there was at first considerable improvement, but on February 26 a purulent discharge began from one ear. No meningococci found in discharge, but abundant streptococci. A subcutaneous abscess appeared in one arm and was opened. The otitis did badly, the mastoid cells being invaded, and a radical mastoid operation was finally performed. Only streptococci were obtained from the mastoid pus. Symptoms of internal hydrocephalus became more pronounced, and on April 4 she was trephined and the ventricle drained. She died, however, on April 5. There was firm matting of the membranes about the base of the brain, and moderate hydrocephalus. No pus was present within the cranium. There was a streptococcal septicæmia. On February 8 the pharynx yielded 90 per cent. of meningococci, but by March 9 the pharynx was clear. Blood culture, forty-eight hours from onset, was negative. Both spinal and pharyngeal strains proved to be Type II.

Case V.—Female, aged 3. Onset February 15; admitted February 17. Lumbar puncture on February 17 and 18. On February 18 she had 15 c.c. new Lister serum intrathecally, and a further 15 c.c. intravenously on February 19, on which day a hæmorrhagic rash appeared. She died on the afternoon of February 19. The meningitis was found to be mainly vertical. One colony of the meningococcus was recovered from the heart's blood, although the body had been frozen forty-eight hours. On February 18 the pharynx yielded about 5 per cent. colonies of meningococci, and these, like the spinal strain, agglutinated with Type II serum alone, but not at all vigorously. The blood strain agglutinated with the same serum and equally weakly.

Case VI.—Male, aged 24, munition worker. Onset February 17; admitted same night, when 80 c.c. of fluid escaped on lumbar puncture. Next day 100 c.c. were drawn off, and 30 c.c. new Lister serum given. Convulsion during recovery from anæsthetic. The case appeared a severe one; the patient was unconscious on admission and had a marked rash. Recovery was immediate, though he had only the single dose of serum; he was convalescent in two days. The pharynx on February 18 yielded 1 per cent. meningococci. On March 16, and probably before, the pharynx was free. Both spinal and pharyngeal strains belonged to Type II.

Case VII.—Male, aged 5 months. Onset March 8. Admitted March 9. Not apparently a very severe case. No rash. Lumbar puncture was done frequently, first on the day of admission, when 8 c.c. of new Lister serum was

given. Further doses of 5, 7, 6, and 10 c.c. were given at intervals up to March 29. The meningococcus was still recovered from the cerebrospinal fluid on March 18. All this time there was marked cervical retraction, but the temperature and pulse gradually fell to normal by March 29. The child then progressed well, and left the hospital on April 20 completely recovered. On admission, the pharynx yielded some 5 per cent. of meningococci. Both spinal and pharyngeal strains were of Type II.

Case VIII.—Male, aged 7. Onset March 9. Admitted March 10. Symptoms not severe, but rash well marked. Nevertheless blood culture (5 c.c.) on March 10 yielded growth of the meningococcus in all six culture tubes. Lumbar puncture twice on day of admission; the second time he had 5 c.c. new Lister serum intrathecally and 10 c.c. intravenously. Another 10 c.c. serum given March 13, by which date the temperature was normal and the cervical retraction had gone, though living meningococci were still present in the cerebrospinal fluid. By March 15 the boy was practically well. On admission, 60 per cent. of the pharyngeal colonies were meningococci, and they were still abundant in the pharynx on March 27. The strains from the cerebrospinal fluid, blood, and pharynx were tested, and were found to be Type II, but were agglutinated by only one of the three Type II sera used, and this only up to 1 in 100 dilution.

Case IX.—Female, aged 17. Onset, with rigor and fever, March 27. Admitted March 28. Collapsed, unconscious and almost moribund, with a temperature of 95° F. Blood culture negative. Lumbar puncture done several times. On March 29 she had 30 c.c. new Lister serum intrathecally and 30 c.c. intravenously, as a scanty petechial rash was appearing. By March 31 she was nearly well, and convalescence was uninterrupted. The pharynx, on admission, yielded a few colonies of meningococci. On April 26 they were still abundant in the pharynx (60 per cent.); these, and the spinal strain, were of Type II.

Case X.—Female, aged 7 months. Date of onset uncertain, probably about March 21. Admitted March 23. No diagnosis till March 31, when lumbar puncture was first performed. Serum was given, 6 c.c. on April 1 and 6 c.c. on April 4, but meningococci were still present in the cerebrospinal fluid on April 16, though the fluid was almost clear. There was great cervical retraction, and signs of internal hydrocephalus developed. Very large quantities of fluid, for an infant, continued to escape on lumbar puncture—e.g., 75 c.c. on April 16 and 85 c.c. on April 19. The ventricle was twice tapped through the anterior fontanelle. The child died on April 20. The ventricles were greatly distended with clear fluid, and the pons and the surrounding structures were plastered with purulent lymph, but there was little or none about the foramen of Magendie. The meningococcus was not found in the pharynx till a third attempt, some days after admission. It proved, like the spinal strain, to be Type II.

Case XI.—Male, aged 8 months. Date of onset probably March 7. Admitted March 17 with cervical retraction, which later became extreme. There was no improvement in spite of repeated lumbar puncture and four doses of the new Lister serum (10, 8, 8, and 15 c.c.), and 6 c.c. intravenously on April 5. The child finally died on April 20, with very great distension of the ventricles (three-quarters of a pint escaped at the post-mortem). The meningitis was almost entirely basal. The meningococcus was readily recovered from the pharynx on admission, but both the pharyngeal and spinal strains, in my hands and in Lieutenant-Colonel Gordon's, refused to agglutinate with any serum, and could not be referred to any known epidemic type.

(May 2, 1916.)

The Treatment of Wound Infections.

By W. PARRY MORGAN.

ATTENTION is being drawn at the present time to the study of the physiological aspect of the treatment of wound infections, and, as a result, new methods, to replace old, have been suggested. In particular, Sir Almroth Wright advocates very strongly giving up treatment by antiseptics for treatment by salt solutions. He does this chiefly on the ground of laboratory experiments, laying it down in respect to treatment that "where the results are neither brilliantly successful nor the reverse . . . we shall be well advised if we guide ourselves, when this is unambiguous, by the verdict of laboratory experiments." Although I have been working with him, I am unable to agree that his experiments conform to this standard, and would for that reason put forward points which appear worthy of further consideration. These relate to: (1) Measures which affect the flow of lymph; (2) measures which affect the flow of pus; (3) antiseptics.

(1) MEASURES WHICH AFFECT THE FLOW OF LYMPH.

In his first lecture [1] on the treatment of wounds, Sir Almroth Wright maintained "that we have at disposition an agency for powerfully increasing the outflow of lymph," and asserted that a 5 per cent.

solution of common salt "brings into play osmotic forces, and 'draws' the lymph out of the walls of a wound by a *vis a fronte*." In his second lecture [2], as a result of the criticism that osmotic forces would determine the flow of water and not of lymph, he appeared to give up the notion of osmosis, but otherwise maintained his position. At this lecture he showed four experiments to "bring clearly before the eye the drawing power of salt." For the first, he took a capillary tube open at both ends, with an additional opening at its middle. This tube he filled with strong salt solution, and, holding it horizontally, dipped it for a few seconds into a watery solution of methylene blue. On taking the tube out, he found that more methylene blue had passed in through the three openings than had done so in the case of a control tube which contained water instead of salt solution. This result we should expect, since salt solution is heavier than water. The salt solution would, for this reason, tend to fall out of the tube, and consequently the methylene blue solution would run in to take its place. The same explanation holds in the case of the second experiment, which was as follows: A piece of glass tubing, plugged with moist cotton-wool, was placed vertically in a vessel containing a watery solution of methylene blue. It was found that the methylene blue travelled up the tube, but travelled higher when salt was placed on the top of the plug. In the third experiment, Sir Almoth Wright took a "stab" of watery agar, and on the top placed a cube of salt. In some twelve hours an interchange had taken place, salt passing into the agar and water to the salt. This he gave as a demonstration of what he expected—namely, "a process of barter in which salt and water should be exchanged, not in volumetric equivalents, but in the ratio of very many volumes of fluid for one of the solid." As a matter of fact, the volume of fluid in such a case is not greater than the volume of solid, and the experiment is merely a demonstration of the diffusibility of salt in agar. The fourth experiment was similar, but in this case the stab consisted of blood agar. Here the presence of the albuminous substances in the fluid which collected above the agar showed only that such substances diffuse in agar in the same way as salt, and not that salt draws lymph. It is therefore clear that these experiments do not support the view that a hypertonic salt solution will draw lymph out of a wound.

Again, Sir Almoth Wright, in his discourse, asserted that it was "confusion of thought" which led to the belief "that it is the interposition of the sieve which confers upon salt the power of drawing water to itself." Such an assertion is really a denial of Newton's third

law, which lays it down that action and reaction are equal and opposite. For it follows from this law that salt would be drawn to water by forces equal to any which would draw water to salt. Consequently, unless a sieve supported and held back salt, while not at the same time preventing the passage of water, there could not be such a process of barter as Sir Almroth Wright suggests. It is plain, then, that the only sense in which salt can be said to "draw" water to itself is that recognized when such a process as osmosis takes place. But salt cannot be said to draw lymph to itself even by osmosis, because albuminous substances cannot pass through membranes impermeable to salt. On the contrary, it might be said that since animal membranes are usually permeable to crystalloids and not to colloids an albuminous substance may by osmosis draw salt to itself.

Having seen that salt does not draw lymph by a *vis a fronte*, we may examine how the outflow can be influenced. We shall only deal with those ways which are clearly physical. For these to be operative, we must presume that the lymph spaces and channels, potential and otherwise, are open to the surface, and that a fluid pressure greater than atmospheric tends to drive lymph out through them. The flow will then be at a rate depending on the pressure in the tissues and on the number and size of the channels. As regards the pressure, it is plain that this will vary with the hyperæmia, and so we may expect that any measure which favours hyperæmia will also act as a lymphagogue. Again, as regards the channels, we see that they may be obstructed (1) by dried lymph on the surface blocking the outlets; (2) by the cellular walls coming together and obliterating such channels. The application of any moist dressing will usually remedy the first, and is as a rule sufficient. In this connexion it must not be thought that moist coagulated lymph will necessarily prevent drainage. After all, it is merely a meshwork of fibrin in which the leucocytes can move and function. It is hardly comparable with a membrane, and is pervious unless it becomes blocked by pus cells. The obstruction due to this and to the obliteration of the channels can be relieved if we can abstract fluid from the cells. This can be done by the application of hypertonic solutions, which cause an osmotic diffusion of water out of the cells. The cells then shrink and the obliterated channels open up, just as the ground is fissured in dry weather. The result is that the lymph has free passage to the surface and the lymph-bound condition is relieved. This action of hypertonic solutions can readily be demonstrated by trying to filter fluids through agar containing blood corpuscles. Owing to the

osmotic crenation and shrinkage of the corpuscles which they cause, hypertonic solutions go through comparatively readily, while the isotonic are held back.

But it is hardly to be expected that the direct osmotic effect of salt in a wound will be more than a very superficial one, for, once a flow is established, the current of lymph outwards will tend to counteract the diffusion of salt inwards. Thus, as soon as the surface becomes pervious, the presence of hypertonic solutions cannot be expected still further to increase the flow. Again, it does not necessarily happen that the osmotic effect of hypertonic solutions will always produce conditions favourable to a flow. From a clinical point of view, this is made clear by Colonel H. M. W. Gray [3], who, at Rouen, in a lecture strongly advocating the use of hypertonic salt solution as a lymphagogue, gave away much of his case by pointing out that salt "tabloid" packs, so far from producing a flow, frequently became dry after twenty-four hours.

It may be observed here that the effect of hypotonic fluids, such as tap-water, hydrogen peroxide, and iodine solutions, have the opposite effect to hypertonic fluids, and would cause the surface cells to swell. This swelling may be expected to bring about a diminished flow of lymph. It is therefore desirable that the excipient used for antiseptics should be isotonic with the blood fluids. For this reason we ought to dilute antiseptics with physiological saline, unless they are incompatible, and not, as is usually done, with water.

(2) MEASURES WHICH AFFECT THE FLOW OF PUS.

Here, again, Sir Almroth Wright claims that he has a specific, this time in physiological saline, asserting that "the white corpuscles are carried forward by a chemotactic movement in the direction of the free surface upon which the physiological solution has been imposed." I would venture, however, to examine in fuller detail the experiments on which he bases this claim, and whether it is warranted by them.

Taking flat capillary tubes containing unclotted blood, he centrifugalized this before it had time to clot. When clotting had taken place there was then in the tube a clot consisting of a red corpuscular portion (fig. 1, *a*), a white plasma portion (fig. 1, *c*), and between these, joining them, a narrow portion containing white corpuscles (fig. 1, *b*). He then superimposed upon these clots a "chemotactic agent," such as saline, and incubated them. The clots were afterwards removed from the tubes, mounted on slides, and stained. It was observed in

many cases that, if the temperature was in the neighbourhood of body temperature, the leucocytes had wandered or emigrated into the white clot. It was on experiments of this nature that Sir Almroth Wright based his statements regarding chemotaxis.

We may get help in understanding the behaviour of the leucocytes in these experiments if we consider the ways in which the clot tends to contract, and, for this purpose, it is what happens in the neighbourhood of the leucocytic layer of the clot which is of importance, as once the leucocytes have moved well out into the white clot they appear to remain there. Sometimes, as Sir Watson Cheyne points out [4], there is no contraction (fig. 1, A). At other times contraction takes place at the junction of the red and white clots (fig. 1, B, C), and a neck containing most of the leucocytes is formed. This neck may lie wholly free in serum (fig. 1, B), or partially in contact with the walls of the tube (fig. 1, C). When it lies wholly free in serum, it becomes highly probable that the wandering movements of the leucocytes will result in their finding a way laterally out of the clot into the serum more readily than along the axis, and thus we would expect to find no emigration in such a case. On the other hand, when the neck of the clot lies partly in contact with the tube, the leucocytes in that situation would not be able to pass out of the clot, and so they would be more likely to travel along the axis and show emigration. These expectations accord with what is actually seen to take place. In my own experiments I found in some cases practically no polynuclear cells in the white clot and only a few mononuclear near the neck. This occurred both with and without the addition of the chemotactic agent. In other cases the leucocytes travelled along the clot, especially the more active polynuclear. This also occurred both with and without the addition of the chemotactic agent. Some of the specimens—specimens showing ultimately no emigration, and to which no chemotactic agent had been applied—I examined from time to time on a warm stage to watch the movements of the leucocytes. At first, near the neck, they could be seen moving actively in all directions, but later they disappeared out of the clot. When mounting these specimens, I collected the serum which had exuded from the clot, and found a very large number of leucocytes in the exudate. Subsequent examination of the tubes, which, after the clot had been removed, were immediately filled with fresh serum, also revealed large numbers of leucocytes adhering to the walls and showing active movements. Hence the polynuclear leucocytes, without the application of a chemotactic agent, do wander into the clot and then

out into the serum. It is clear, therefore, that Sir Almroth Wright can hardly be justified in supposing that emigration due to wandering or "eleutherotropic" movement is predominantly mononuclear and that polynuclear emigration is chemotactic. This variable loss of leucocytes from the clot to the serum, clearly independent of any possible chemotactic influence, shows that it is as futile to aim at measuring quantitatively the emigration movements and chemotactic effects by estimating the number of leucocytes which remain in the white clot at different distances from the red as it would be to estimate the total number of a company after an unknown proportion had retired. But

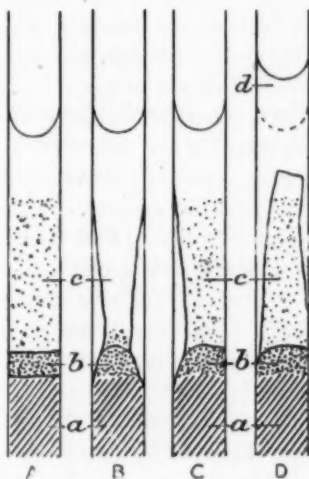


FIG. 1.

Emigration tubes showing different types of contraction. (a) Red clot, (b) leucocytic layer, (c) white clot, (d) superimposed fluid. (A) No contraction; free emigration shown. (B) Contraction with the formation of a neck at the leucocytic portion of the clot, which lies free in serum; emigration is not shown in this. (C) Contraction similar to (B), but the neck is partly in contact with the tube; emigration shown. (D) Chemotactic agent superimposed; retraction of clot along its axis; leucocytic layer in contact with the tube; emigration shown; this clot is not so likely to contract in mounting, so that the distance which leucocytes traverse would appear greater than in those from (A) and (C).

even when free emigration takes place in the presence of physiological saline it cannot be said to be due to chemotaxis, for the distance traversed by the leucocytes is little or no greater than when no fluid

but serum is present, whereas if such emigration were due to chemotaxis—that is, “a directed movement along a particular axis undertaken under the direction of a chemical stimulus”—one would expect great differences. The small differences that are occasionally seen may be explained by a slight effect which physiological saline may possibly have in increasing the non-directed wandering movements of the leucocytes. This effect is apparently shown in some specimens obtained by mixing saline with blood before it clots, circumstances in which there could be no chemotaxis. But even admitting this stimulation, it is doubtful whether it is of any value. For the leucocytes would not start their wanderings in large numbers, but only travel faster through the infected tissues, and act there for a shorter time. Again, physiological saline would not increase the all-important function of phagocytosis. I submit, therefore, that laboratory results, as yet, give no grounds for asserting that we have in physiological saline an agent which will advantageously affect the activities of the leucocytes and the flow of pus into a wound.

On the other hand, there is no difficulty in agreeing that strong antiseptics, bacterial suspensions, and, above all, hypertonic solutions do act in suppressing leucocytic movements. This is clearly shown by experiments made by mixing the blood and the test fluid in the emigration tubes before clotting takes place. Such experiments show that some antiseptics—such as carbolic acid—powerfully suppress emigration, whilst others—such as mercuric salts and the neutral hypochlorous solutions—have comparatively little action. But of more practical significance in determining the possible injurious effects produced by the various applications on the physiological processes is the observation made on the wound itself as to whether there is a good flow of laudable pus. If there is, we may be sure that the application is not doing much harm to the tissues, and that the physiological processes remain active. In this connexion it may be noted that Sir Almroth Wright points out “that the practitioner of to-day has been educated up to expect to find, within a few hours after washing out an infected wound with antiseptics, as much pus as when he last came to dress it”; and that a hypertonic salt solution will, as long as its concentration is maintained, arrest all suppurative processes and “give us a wound as clean and as free from pus as meat.” The inevitable conclusion from this is, that in practice, treatment by hypertonic saline, by interfering with the activities of the leucocytes, renders ineffective the only defence there is against the streptococcus and the staphylococcus, whilst, with the application of an antiseptic or of physiological saline, this defence comes again into action.

But some do not view with satisfaction a good flow of pus from a badly infected wound, and it is not uncommon to hear the contemptuous expression "pus poultice." Certainly if the pus is stale and has become "corrupted," it is noxious, but if fresh, it is efficiently doing its work of combating the organisms. Our aim must therefore be to remove the pus before it becomes corrupted, and if possible to destroy the organisms which corrupt it. The methods of removing pus are, of course, by constant irrigation and occasional flushing, and in deciding between these it must be remembered that the use of irrigating fluids, even of physiological saline, if delivered below body temperature, will do harm by retarding the activities of the leucocytes. The possibility of destroying the organisms brings us to the consideration of antiseptics.

(3) ANTISEPTICS.

The attempt to destroy organisms in wounds is made in two ways: (1) by washing out with lotions; (2) by the application of dressings. In connexion with the washing out of a wound, it will be recognized that, although, as has been observed, the greater part of the wash will be thrown down the sink, a residue will remain in the recesses and pockets; and also that even on the most exposed surfaces some lotion will remain, wetting the films of pus adhering to those surfaces. These will continue to be a source of infection unless they can be sterilized. We have thus to consider the effect of antiseptics on them. In the case of the residue, there is a comparatively large quantity of antiseptic mixed with a small amount of pus, and for the purpose of estimating the effect of the antiseptics, I took it that the conditions of the following experiment were analogous.

Mixtures of one part of pus and nine of antiseptic were made, and, after ten minutes, 10 c.mm. planted in tubes of liquid agar at 42° C. After they were thoroughly shaken, the tubes were sloped and the agar allowed to set. They were then incubated at 37° C., and after a period were examined for colonies. It was found that when the antiseptic was strong the number of colonies could be counted readily. When it was weak, and in the case of the controls, the agar became opaque with innumerable minute colonies. The results were comparative, and did not lend themselves to strict quantitative expression. This was to be expected, for the specimens differed considerably. All were heavily but not equally infected. Some were thick and mucoid, and would not mix well with the antiseptics. However, in the case of every sample of pus, several antiseptics and a control were tested.

I give a table (I) to illustrate the kind of result obtained. Using this method, I came to the following conclusions: For the purpose of washing out a wound, neutral hypochlorous solutions are by far the most potent of the antiseptics usually employed, and are effective if diluted to 1 in 800 available chlorine, or 1 in 4 of the strengths usually dispensed. Mercury antiseptics 1 in 1,000, and

TABLE I.—SHOWING THE EFFECTS OF ANTISEPTICS UPON BACTERIA IN PUS.

<i>Experiment I.</i>					
Iodine :—	1 : 400	1 : 800	1 : 1,600	1 : 3,200	1 : 6,400
16 hours ...	+	++	+++	+++	+++ gas
Biniodide of mercury :—					
16 hours ...	+	++	++	+++	...
40 hours ...	++	+++	+++	+++	...
Carbolic acid :—	1 : 40	1 : 80	1 : 160	1 : 320	
16 hours ...	+	++	+++
40 hours ...	++	+++	+++
<i>Antiseptic diluted with hydrogen peroxide (5 vol.).</i>					
Biniodide of mercury :—	1 : 400	1 : 800	1 : 1,600	1 : 3,200	1 : 6,400
20 hours ...	—	—	+	+++	+++
40 hours ...	—	—	++	+++	+++
Carbolic acid :—	1 : 40	1 : 80	1 : 160	1 : 320	
20 hours ...	—	+	++	+++	...
40 hours ...	—	++	+++	+++	...
<i>Experiment II.</i>					
Hypochlorous solutions...	1 : 1	1 : 2	1 : 4	1 : 8	
Available chlorine ...	1 : 200	1 : 400	1 : 800	1 : 1,600	
Dakin's solution :—					
18 hours ...	—	—	—	+	
3 days ...	—	—	—	+	
Eusol :—					
18 hours ...	—	—	—	+	
3 days ...	—*	—	—	++	
Carbolic acid ...	1 : 80	1 : 160	Control	...	
	++	+++	+++	...	

carbolic acid 1 in 80, never sterilize completely, but kill a very large number of microbes, and delay the appearance of the growth of those which remain. Hydrogen peroxide by itself has very little bactericidal power, but when added to other antiseptics with which it is not incompatible, it increases their bactericidal power on pus organisms, presumably

by its mechanical effect. Most of the other organisms in pus are killed more surely than streptococci and staphylococci, an action which is distinctly more marked in the case of mercury and carbolic acid than in the case of iodine and hypochlorous solutions.

In the case of the film of pus which the mechanical disturbance of washing does not remove, we again have a large quantity of antiseptic acting for a comparatively short time. I considered here that the effect of the antiseptic could be judged by the following experiment: 20 c.mm. of pus were smeared on the surface of agar slopes and allowed to dry. The tubes were then filled with antiseptic lotion so that the films were completely covered. After an interval the antiseptic was poured away and the tubes incubated upside down, so that any excess drained away. The following table (II) illustrates the results:—

TABLE II.—SHOWING THE EFFECTS OF ANTISEPTICS UPON BACTERIA IN FILMS OF PUS.

Antiseptic	Strength	Time	
<i>Experiment I.</i>			
Carbolic acid	1:80	9 min.	Growth as in control
Eusol (1:200 available chlorine)	1:4	1½ "	All but a few small areas sterilized
" " "	1:4	3 "	" " "
" " "	1:4	5 "	" " "
" " "	1:4	10 "	All but one small area sterilized
" " "	1:8	2 "	Two small sterile areas
" " "	1:8	5 "	A few sterile areas
<i>Experiment II.</i>			
Carbolic acid	1:80	11 min.	Many discrete colonies all over film
" " "	1:80	40 "	Sterile
Iodine	1:1,000	4 "	"
Binioidide of mercury ...	1:1,000	4 "	"
Hydrogen peroxide	2·5 vol.	10 "	"
Dakin's solution (1:200 available chlorine)	Undiluted	1½ "	All but a small area sterilized
" " "	" "	10 "	Sterile
" " "	1:2	5 "	Two colonies
" " "	1:2	10 "	" "
" " "	1:4	5 "	All but a few small areas sterile
" " "	1:4	10 "	All but one small area sterile
" " "	1:8	10 "	Good growth; part of film sterile
Control			Massive growth

The results are very similar to those of the first experiment. One point was clearly shown, that the thickness of film makes much difference to the efficacy of the antiseptic. It was clear also that some

antiseptics were much more rapid in their action than others, notably the hypochlorous solutions. This suggests that these are the most suitable for the purpose of washing out a wound, especially as they have practically no effect on the activity of the leucocytes.

In the case of the dressings, the antiseptic is gradually diluted with pus, becoming at the same time less efficient. Here, therefore, the pus tends to be in excess. To test the effect of antiseptics in these circumstances, I took either a specimen of pus containing very few organisms, one or two per field of a film, or a very thick suspension of blood corpuscles in serum containing a few organisms, and added a proportion of antiseptic. A film was prepared from the mixture, which was then incubated. After a time another film was prepared and compared with the first. If no difference in the number of organisms before and after could be detected, I concluded that the growth was prevented by the antiseptic. If there was a difference, it is clear that the organisms grew in spite of the presence of the antiseptic. A mixture of pus and saline served as control. In the table (III) which I have set out, the strength of the antiseptics given is the ultimate strength in the mixtures. Thus a strength of biniodide of mercury 1 in 1,000 would be present in a mixture of four parts of pus and one part of 1 in 200 of the antiseptic. In these experiments I found it made very much difference whether phagocytosis had taken place or not, there being an advantage in favour of the antiseptic when it had not—that is, organisms grew better (*a*) in pus from an infected wound than in sterile pus to which a suspension of microbes was added; (*b*) in the infected blood when it had been incubated so as to allow phagocytosis to take place. This would lead us to expect that antiseptic dressings would be especially useful (*a*) in the earlier stages of an infection, before there is much pus; (*b*) to prevent the growth of organisms which might obtain access to a wound during the dressing. The following are the conclusions which I consider these experiments justify: (1) The ultimate strength of the antiseptic being the same, the greater the proportion of pus the less the inhibition of growth. (2) Where pus is present in the proportion of 4 parts to 1 of antiseptic, organisms may grow freely when the following are the antiseptics used: Mercuric salts 1 in 200, carbolic acid 1 in 60, iodine 1 in 200, boracic acid 1 in 20, chloramine 1 in 20, hypochlorous solution 1 in 200 available chlorine. Organisms also grow when salt solution 20 per cent. is used. (3) Since organisms grow in pus in which antiseptics are present in the strengths indicated above, it is utterly unreasonable to expect any of them to diffuse into the tissues to

such an extent as to give a strength sufficient to inhibit the growth of microbes, still less to kill them. (4) The pyogenic organisms are, as regard their growth in pus, among the least affected by the ordinary antiseptics.

TABLE III.—SHOWING THE EFFECTS OF ANTISEPTICS UPON THE GROWTH OF BACTERIA IN PUS.

Experiment I (4 parts of pus, 1 part of antiseptic).

Carbolic acid	1 : 240—	...	1 : 480+
Hydrogen peroxide (10 vol.)	1 : 5+
Iodine	1 : 1,000—	...	1 : 2,000+
Biniodide of mercury	1 : 1,000+

Experiment II.

Iodine :—

3 parts antiseptic, 1 part pus	...	1 : 1,600—	...	1 : 3,200+
1 part " 1 " "	...	1 : 1,600—	...	
1 " " 3 parts pus	1 : 1,600+
1 " " 4 " "	1 : 1,000+

Biniodide of mercury :—

3 parts antiseptic, 1 part pus	...	1 : 800—	...	1 : 1,600+
1 part " 1 " "	...	1 : 900—	...	1 : 1,400+
1 " " 3 parts pus	...	1 : 800—	...	1 : 1,600+
1 " " 4 " "	1 : 1,000+

Experiment III (4 parts blood corpuscles with staphylococcus-infected leucocytes, 1 part antiseptic).

Carbolic acid	1 : 200—*	...	1 : 300+
Chloramine	1 : 75—*	...	1 : 100+

Experiment IV (1 part blood corpuscles, 1 part antiseptic).

Hypochlorous solutions (1 : 200 available chlorine) :—

Dakin's solution	...	1 : 2 (?)†	...	1 : 4+	...	1 : 8+
Eusol	...	1 : 2—†	...	1 : 4—†	...	1 : 8+
Carbolic acid	...	1 : 160—*	...	1 : 320+		

* Subcultures on agar gave no growth.

† Subcultures on agar gave a little growth.

The results obtained with hypochlorous solutions are of particular interest at present, inasmuch as eusol is being advocated for intravenous injection in cases of septicæmia. If they have any beneficial effect in the blood, it is clear the explanation cannot be found in direct bactericidal action.

When these experiments are reviewed as a whole, it becomes plain that in appraising the value of an antiseptic the purpose for which it is used must be taken into account. For instance, for washing out of a

wound, when the antiseptic would be in great excess, hypochlorous solutions are very potent and carbolic acid is comparatively weak. On the other hand, for an application in a dressing, when the pus would tend to be in excess, hypochlorous solutions are practically useless, whilst carbolic acid, although it has the disadvantage of interfering with the activity of the leucocytes, is fairly efficient.

Again, it becomes clear that, upon the evidence the experiments supply, antiseptics cannot do as much as is claimed for them. They certainly cannot sterilize the tissues subjacent to the surface of a wound, and, indeed, cannot be depended upon to sterilize an accessible surface, although they kill many of the organisms on it. Their use, therefore, depends on whether there is any advantage in this. It would seem that there is, for it can hardly be possible that the depth and intensity of the tissue infection are independent of the proportion and virulence of the organisms in the surface pus. If, therefore, these can be reduced, even temporarily, without at the same time unduly interfering with physiological processes, it is an important gain. This, which is in fact the most important object in dressing wounds, can be done by means of antiseptics, and herein lies their rôle in antiseptic treatment. If we expect more from the antiseptics at present available, we shall be disappointed.

In conclusion, I would express my grateful acknowledgments to those with whom I have been associated under the auspices of the Army Medical Service and of the Medical Research Committee; but in particular to my former chief, Colonel Sir Almroth Wright, although I have come to conclusions divergent from many of those at which he has arrived.

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DISCUSSION.

Dr. D'ESTE EMERY: I regret that the lateness of the hour prevents my dealing adequately with Dr. Parry Morgan's paper, which teems with controversial points, on many of which I am in strong disagreement with the author. As regards the first question, the power of hypertonic salt solution to cause the flow of a large quantity of lymph from a wound, Dr. Parry Morgan appears to object both to the idea that it is a process of osmosis and also to the simpler and non-committal expression that the fluid is drawn out by the salt. The point is one that might properly be discussed before a physical society, but the important practical thing is that the application of a hypertonic solution to a sloughy wound the walls of which are teeming with organisms may determine a copious flow of what is, if not lymph, at least a fluid containing protein substances similar to those of lymph, and, what is of supreme value, containing defensive substances, so that after twenty-four or forty-eight hours no organism at all can be found microscopically in the secretion from the walls of the wound, though they may still be present in small numbers in cultures. I have had a difficulty in following Dr. Parry Morgan's account of how this flow of fluid is brought about, and am not familiar with the channels with cellular boundaries about which he speaks as existing in the walls of wounds. Further, if such be present I should have thought that if the tissues composing them are caused to shrink by the application of a strong hypertonic solution they would be narrowed and the outflow of fluid obstructed. Secondly, with regard to the formation of pus, I consider that the author somewhat misrepresents the views of the physiological school of treatment. The work of the leucocytes is done in passing through the infected tissues forming the walls of the wound. In their passage they ingest all the bacteria they can, and then pass on outward into the lumen, where they appear as pus. This is in itself a bad thing, and must be got rid of by irrigation or drainage just as soon as possible; but it is an indication that the leucocytes are actively at work clearing out the bacteria from the tissues, and, as such, is a good sign. Whatever may be the technical difficulties in the way of investigation on this subject *in vitro* (and I think they may be overcome), I would point out that there is no doubt that in actual clinical practice the application of normal salt solution to the walls of a wound which has been treated with hypertonic salt solution does, as a matter of fact, determine such a flow of leucocytes to the surface. Dr. Parry Morgan appears to agree with Sir Almroth Wright that salt in certain dilutions does stimulate the activity of the leucocytes, but he does not think this is any advantage. I disagree with this view, and would point out that the more rapidly the leucocytes travel in the infected tissues the more quickly and thoroughly will they remove any bacteria which they encounter. On the third question, that of antiseptics, I agree with the author that it is hopeless to expect them to diffuse any reasonable depth into the tissues in such amounts as to kill the

organisms. The author's views mainly apply to the parts at a fairly late stage, when granulation tissue is present. At this stage perhaps not much harm is done by antiseptics, but the case is different in the early stages. At this time the walls of the wound are formed of bruised and damaged tissues, sometimes inches thick, which are so injured as to be just on the borderland between life and death. A very small extra injury may be sufficient to cause much greater loss of tissue than will be the case if all the conditions have been made favourable, and any antiseptic, whatever its nature, which will kill bacteria will kill healthy tissues, and, still more, damaged tissues, even though it be more highly diluted. It appears, therefore, that the use of strong antiseptic lotions in the early stages of wound treatment may be a source of grave danger.

Dr. PARRY MORGAN: I am glad to hear that Dr. Emery justifies the use of hypertonic solutions on clinical grounds rather than on laboratory experiments. Dr. Emery does not make it clear how I have misrepresented the views of the physiological school. Dr. Emery's statement that normal saline solutions determine the flow of pus into wounds previously treated with hypertonic solutions somewhat misrepresents the facts. It is the removal of the strong salt solution which is the determining factor, and in practice it matters not whether normal saline or an antiseptic is used for the purpose, the cells resume their activities. In regard to the objection that antiseptics will injure the tissues, may I observe that the same thing must be said of hypertonic solutions.

Pathological Section.

President—Dr. F. W. ANDREWES, F.R.S.

(May 2, 1916.)

The Traumatic Causation of Appendicitis.

An Examination of the Contents of 100 Vermiform Appendices taken consecutively from Persons dying at and over the age of 40; and of the Contents of Twenty-five Appendices taken from those dying between the ages of 15 and 25 inclusively; together with an Examination of a Series of Appendicular Concretions, and Observations upon Appendicular Pigmentation; &c.

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Contra originem appendicitis traumaticam.

SUMMARIUM.

DATAE sunt hae observationes quibus probarentur sententiae quaedam de appendicitis origine hic et illic propositae.

Causarum quae proponuntur traumaticarum una est vitrum a vasibus ferreis desquamatum.

Hujus vitri nunquam inveni squamas aut in appendicibus normalibus (centum et viginti quinque) aut in concretionibus mediis (viginti) inclusas.

De laesionibus silice glutito forsitan causatis, appendicitis, ut didici, paene incognita est in operariis qui apud Africam australem, in metallis laborant.

Magnesii silicatis (cretae gallicae) quae pulveribus ad dentes fricandos admisceri solet, nihil, iterum, inveni aut in concretionibus aut in appendicibus normalibus inclusum.

De chalybis, praecipue, fragmentis abrais a cylindris quibus in farinâ tritendâ nunc ubique pistores utuntur, observationes meae, mutatis mutandis, idem demonstrant.

Appendicis nigritiem nonnunquam in organis excisis observatam, intussuptioni chalybis in farinâ inclusi, non ascribere possumus, quum sphaerulae fuscae quae in cellulis membranae mucosae includuntur, nullas ferri notas microscopicas monstrent.

Exemplis paucis exclusis, ab appendicis concretionibus nuclei absunt.

Concretio vulgaris ex herbarum residuis solum constat, calcii carbonate et phosphate, in gradibus diversis, commixtis.

Nullam inveni concretionem de pilis avenae constructam, ut illae quae in colo repertae sunt.

Inter se vix delimitantur appendicis scybala et concretiones laminatae. Concretionis laminatae, quidem, nucleus scybalis minutis aggregatis nonnunquam repraesentatur.

Concludi ergo debet:—

(1) Appendicitem particularum ingressu irritantium, in cibo inclusarum, non causari.

(2) Concretiones appendiculares circum particulas inorganicas intra appendicem cumulas non formari.

THE observations indicated in the title of this communication were undertaken with the object of testing certain views which have been put forward in explanation of the alleged increase of appendicitis.¹ The views in question suppose a traumatic as a predisposing cause, and assume that the ingress of inorganic material to the appendix is an important factor in the genesis of the disease. In searching for such a cause, one must bear in mind that certain conditions in regard to it, have to be fulfilled:—

(1) The factor, whatever it may be, must not only be widespread, but it must extend to all classes of the community, if it is to be commensurate with the clinical data.

(2) It must be one of comparatively recent introduction.

I may first deal with the suggestions which have been put forward in this direction, discuss the possibilities theoretically attaching to them, and finally give the actual results of a systematic investigation, and see

¹ This increase is not universally held by surgeons of equal standing and experience. It might be here suggested with due deference that the rise and decline of disease would form a proper subject of study for the Section of the History of Medicine; and that this particular question would be an excellent one to debate.

how far the suggestions are borne out by them. The chief, then, which have been made are:—

(a) That the foreign material comes from the enamelled hardware now so extensively used as cooking and other domestic utensils; and most must be familiar with the manner in which the enamel tends to scale off from vessels of this kind, under wear, especially if they chance to get heated in the dry condition. Such particles, which consist of silica fused into a glaze, are not only insoluble in the gastric and intestinal secretions, but they would be quite capable of inflicting injury upon a mucosa like that of the appendix, if driven upon it by muscular



FIG. 1.

A small scale of saucepan enamel, examined in Farrant's medium.
($\frac{3}{8}$ obj., ocular 4.)

Vitri lamella a vase ferreo desquamata. (Paulum magnificata.)

contraction. Their characters are shown in the accompanying figure, the material having been obtained by heating an enamelled saucepan in the dry state over a Bunsen flame.

In a microscopic preparation examined in Farrant's medium, the particles appear as highly irregular, sharply edged, translucent flakes or scales, which exhibit minute spherical cavities of varying sizes produced from the ebullition of the glaze, their whiteness being due to this cause (fig. 1). If a parallel were drawn between the theoretical possibilities

here opened up, and what is true in regard to pneumoconiosis, it might be inferred that such flakes would be particularly injurious. For of the different materials inhaled into the lung in occupational diseases, silica is the most deleterious. This was the belief of Greenhow,¹ who was the first in this country to describe these forms of pulmonary disease, and to demonstrate the presence of silica in the ash of the affected organ. The injurious effect of inhaled silica has more recently been particularly insisted upon by Dr. E. L. Collis.² Not that other inhaled materials fail to produce fibrosis; but it is silica which renders the lung most prepared for a superadded tubercular infection. Dr. Watkins-Pitchford observes that there is no industrial phthisis amongst operatives who inhale the dust from coal, chalk, plaster of Paris, bricks, tiles, emery, slag-wool, glass, and Portland cement; and that needle and other steel grinders, and flour millers developed their phthisis not from the inhalation of steel or flour, but from the grindstone: the old-fashioned grindstone was made of French buhrstone, millstone grit, or other very hard sandstone, and it was the refacing of the grindstone, or the running of it in a dry condition, which has been in the past so prolific an indirect cause of industrial phthisis. On the mere score of damage the fragments of silica, presumably present in the older stone-milled flour, as contrasted with that milled with steel rollers, would have been more potent for evil than particles of steel, since they are absolutely insoluble in the gastric and intestinal secretions. Certain results following the ingestion of powdered glass are known, from this material having been used for criminal purposes. Unless, however, it is only coarsely pounded nothing harmful ensues under such circumstances; the one case cited in Taylor's "Medical Jurisprudence"³ being that of an infant who was killed by the administration of a quantity of roughly powdered glass, a considerable amount of which was found after death in the stomach, which was coated with a thick layer of tenacious mucus streaked with blood. Dr. A. Mavrogordato tells me that he has recently given finely powdered glass, &c., in food to guinea-pigs without producing any result; the administration extended over a period of one month. Three animals were used: one was given coal dust, one "flue dust,"⁴ and another, powdered flint; in the case of the last, powdered glass was several times substituted:

¹ *Trans. Path. Soc.*, 1866, xvii; and 1869, xx.

² *Public Health*, xxviii, Nos. 11 and 12; xxix, Nos. 1 and 2; 1915, Milroy Lectures.

³ Fifth edition, by Dr. F. J. Smith, 1905, ii, p. 847.

⁴ The dust from the chimneys connected with engines driving mining plant.

no lesions were discovered in the alimentary canal or in the mesenteric glands. In passing, it may be pointed out that no mammals other than man, have a caecal appendix, except apes.

These results confirm the findings of Le Sauvage,¹ who himself swallowed a considerable number of glass particles without experiencing any inconvenience; $2\frac{1}{2}$ dr. were given to a cat without subsequent injury; and 6 oz. or 7 oz. in eight days to a dog, with similar negative results. In India the administration of pounded glass for criminal purposes, homicidal and suicidal, is of well-known occurrence. If it is attended with a fatal result, this must be rare; for a reference to the well-known work² by Dr. Norman Chevers will show that none such is detailed; and, what is still more, in several instances in which glass has been crushed with the teeth and eaten by would-be suicides, mountebanks, and drunkards, either no symptoms have followed, or only temporary inconvenience.

In the more recent publication of Dr. I. B. Lyon,³ one fatal case, in which the cause of death was authenticated, is detailed—viz., that of a man who was attacked with symptoms of irritant poisoning and died in forty-eight hours; the gastric mucosa was reddened, and a quantity of powdered glass was found in the contents of the stomach; no irritant substance other than powdered glass could be found in the viscera.

Whatever it may have been in the past, stone-milled flour is at the present time remarkably free from foreign material. In order to test this I obtained samples from two sources, one from a Wimbledon firm which lays itself out to supply it; the other from a mill in Suffolk. The flour contains the embryo or "germ" as well as the starch, the pericarp or "bran" being removed by sifting. The examination was carried out by irrigating the siftings obtained by means of a triple sieve of brass wire, the sieves being of increasingly fine mesh, the finest having 1,225 perforations in a square centimetre.

Observation 1.—The Wimbledon flour (known as "Stanmylne," an old Saxon equivalent of stone-milled) on being sifted, left a fairly abundant residue of small, white grains on the lowest, finest sieve; these consisted microscopically of aggregated starch grains. On transferring the whole of this residue to a large, flat, glass dish of water, and rubbing it with the finger tips, the material was

¹ "Inaugural Dissertation," Paris, 1820.

² "Medical Jurisprudence of India," 3rd ed., 1870, p. 287.

³ "Medical Jurisprudence for India," 1889, p. 229.

readily broken down, nothing resistant being encountered; and when irrigated, not a particle of silica or other sedimentary residue was left in the dish.

Observation 2.—This was made upon "whole meal," stone-milled flour, supplied by the same firm. On the highest of the three sieves were retained the coarser fragments of the pericarp ("bran"). On the second sieve was retained an abundant debris of pale brown colour, as on the first, but consisting of finer particles of pericarp. On being irrigated, no sedimentary residue of any kind was discovered. On the third sieve was a more abundant material than the preceding, of dirty white colour, which consisted microscopically of groups of starch grains and pertaining cell fragments, pieces of pericarp and a few thick-walled, pointed hairs, with expanded base, derived from the summit of the wheat "berry," or grain. On being irrigated, no sediment of any kind was disclosed.

Observation 3.—This was carried out upon a sample of stone-milled flour from Suffolk, recently ground from English-grown wheat. On the first sieve nothing was retained. On the second there was a thick covering of flour, which consisted microscopically of starch and aleurone grains, fragments of pericarp, and thick-walled hairs derived from the summit of the grain. Irrigation disclosed no silica. On the third and finest sieve the abundant layer of flour had the same microscopic characters; on irrigation no sedimentary silica was found. Of the flour which had passed through the finest sieve the microscopic examination showed that the groups of starch grains were smaller in size; the number of hairs, also, was less; and the hairs were shorter than in the contents of the third sieve. Irrigation of the whole yielded no sedimentary silica.

Even in the case of the inferior flour ground between stones for cattle, no silica reaches the final product in well conducted mills. A sample of this, which I obtained from the large mills of Messrs. Rank of Canning Town, yielded no trace of sedimentary grit on irrigation. Before the passing of the Adulteration Act, 1872, a large amount of indigestible foreign material must have been consumed with food. Flour was adulterated with alum, chalk, bone-dust, and plaster. Dr. Letheby found in certain kinds of tea as much as 40 per cent. of iron filings, and 19 per cent. of silica in the form of fine sand.¹ The alleged increase of appendicitis, however, postdates the Act, and the problem of a traumatic causation is thus reduced to fairly narrow dimensions.

Does the prolonged ingestion of silica induce appendicitis? A convincing answer to this question is furnished by the evidence gained from mining districts. And I may here introduce a report which has

¹ Taylor's "Medical Jurisprudence."

been kindly drawn up for me by Dr. Watkins Pitchford, the present Director of the South African Institute for Medical Research, in Johannesburg :—

"Appendicitis amongst Gold-miners.—In reply to your inquiry of August, 1915, there does not seem to be any undue prevalence of appendicitis amongst miners, as compared with people of other occupations, in this district. Those suffering from miners' phthisis might be expected to give a history of appendicitis, if mine dust were a causal or predisposing agent of the latter disease. Dr. F. Aitken, the Superintendent of the Miners' Phthisis Sanatorium near this town (Johannesburg), tells me that 537 cases of miners' phthisis have passed through his institution, that he has not seen a single case of appendicitis amongst the patients, and has not noted any history of attacks before admission under his care. Dr. H. A. Loesser, the Senior Medical Officer of the Crown Mines, tells me that he has neither noticed, nor heard it suggested, that the disease is more prevalent amongst miners than amongst others. The Director of the Native Affairs Department issues a monthly report dealing with the population, and mortality statistics of the Natives employed on the works and the mines of the Rand. There were 210,247 Natives so employed in July last. Appendicitis is so rare amongst these natives that deaths from such a cause are not classed separately. The Director states that he considers 'mine Natives are not at all liable to the disease.'"

Besides the possibility of mechanical injury, the foreign material, were it to accumulate in the appendix, might serve as the nucleus of a concretion, and become indirectly a cause of appendicitis on the super-addition of an acute infection. Whether this is so or not will appear later.

(b) As a second possibility there is the more widely-spread use of dentifrices and tooth powders. The use of these might involve the daily ingestion of minute amounts of calcium carbonate left in the mouth, or of magnesium silicate (French chalk) which some dentifrices contain in place of the former. The free hydrochloric acid of the gastric secretion would, of course, suffice to convert the first into soluble calcium chloride, and so prevent its reaching the appendix. "French chalk," however, is absolutely insoluble in the alimentary canal. Although an impalpable powder, it consists microscopically of thin, transparent, structureless scales or flakes, as shown in the accompanying figure (fig. 2). Whilst incapable of inflicting mechanical injury, its

accumulation might theoretically serve as the starting point of a concretion, and in this indirect manner become the cause of a subsequent appendicitis.

(c) The extensive use of Cerebos salt is a third suggestion *à propos* of the alleged increase in appendicitis. Cerebos salt contains a marked proportion of insoluble magnesium phosphate. In its preparation the objectionable hygroscopic magnesium chloride present in native salt is got rid of by the action of sodium phosphate; this, of course, leads to the formation of non-hygroscopic magnesium phosphate and a further amount of sodium chloride. As to the fate of magnesium phosphate



FIG. 2.

"French chalk" (magnesium silicate), examined in glycerine. ($\frac{1}{2}$ obj.)

Magnesi silicatis lamellae (cretae Gallicae). (Modice magnificatae.)

in the alimentary canal: if the insoluble residue of Cerebos salt in water is treated with a solution of hydrochloric acid of the percentage in which this is present in the human gastric secretion, and incubated at 37° C., it readily dissolves, with the accompanying formation of magnesium chloride. This possibility may be, therefore, ignored.

A fourth suggestion, which, by reason of its promise and of the attention that has been drawn to it, I propose to discuss at length, is:—

(d) That the foreign material consists of particles worn off from the steel rollers which have now almost universally replaced stone for the grinding of wheat into flour.¹ Assuming that particles of steel are present in flour one must, for the purpose of tracing their fate, determine what would happen to such (1) before the flour was used; (2) during the baking of bread; (3) after their ingestion. The following experiments were carried out in regard to these questions:—

July 26, 1915: A plentiful amount of dry iron filings was shaken up with a sample of stone-milled flour and allowed to stand in a covered glass jar in a living room. Before use the filings (which were obtained from the chemical laboratory of the school) were washed first in ether, then in absolute alcohol, and finally dried. The material was periodically inspected, but without being disturbed. The particles, whether at the exposed surface or in the depth of the flour (as seen against the sides of the glass), remained perfectly free of rusting; and many in the second position brilliantly reflected the light as in the natural condition.

On September 24, 1915, a microscopic preparation, made in glycerine, showed the numerous particles of iron amongst the starch grains to be absolutely unaltered. When viewed by direct light they exhibited a black ground stippled with bright reflecting points, exactly as in a control specimen of the dried filings put up in glycerine for the purpose of immediate comparison. A second examination made on December 4, 1915, by rubbing a small portion of the flour and filings in Farrant's medium, gave the same result, as did similar examinations made on December 20, 1915, and on March 15, 1916. The observation was closed on March 30, 1916, when a considerable number of the particles were isolated by irrigation, cleansed of adherent starch by gentle brushing, and then washed in absolute alcohol. They were all free of rust, and all reflected light as in the natural state. On microscopic examination by direct light, in Farrant's medium, they sparkled with fine points and lines in the characteristic way.

It may be mentioned, as a technical detail, that starch grains themselves, when viewed by direct light in Farrant's medium on a dark ground, are brilliantly reflective. Any possibility of error arising from such a cause is eliminated by washing, since any grains that still adhere to the metallic particles then cease to exhibit this character, when examined in a fluid medium; the brilliancy and fineness of the stippling of the iron, moreover, exceed that of the starch.

December 14, 1915: The preceding observation was exactly repeated, substituting steel filings for the iron. The filings were washed first in ether and afterwards in absolute alcohol, dried, and mixed with stone-milled flour. The mixture was placed in a glass jar, the mouth of which was covered with

¹ W. H. Battle, *Lancet*, 1913, ii, p. 135; *Proc. Roy. Soc. Med.*, 1915, viii (Surg. Sect.), p. 26.



FIG. 3.

A particle from an unused sample of the iron filings referred to, examined in glycerine by direct light, showing the bright metallic stippling and parallel ridges due to the action of the file. ($\frac{1}{8}$ obj.)

Ferri scobis particula una ; eminentia ejus quaeque punctis minutis scintillat.

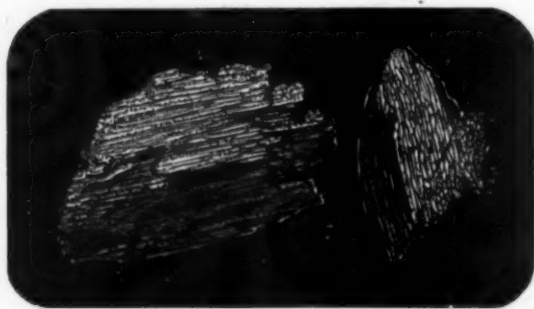


FIG. 4.

Two particles from the steel filings of the kind used to mix with the flour, as described in the text, examined by direct light in Farrant's medium, showing the areas of scintillating points and brightly punctated parallel ridges resulting from the action of the file. ($\frac{1}{3}$ in.)

Chalybis scobis particulae duae : lineis atque punctis scintillant.

filter paper, and kept in a living room until March 30, 1916. As in the preceding case, the particles, whether at the free surface or as viewed at the sides against the glass, underwent no rusting, but appeared black or bright according to the incidence of light. On March 30, 1916, a collection was isolated by irrigation, lightly brushed to remove adherent starch grains, and critically examined; to the naked eye the filings were, without exception, quite bright as when first mixed with the flour.

October 26, 1915: Finally, a similar observation was made with needles, which were dried after washing in ether and absolute alcohol. The needles were buried in the flour on October 26, 1915, and examined at intervals by withdrawing some with dry forceps. They remained quite bright for five months, when the observation was closed.

It will appear, therefore, from these data that particles of steel detached from fluted rollers would retain their characters for an indefinite period in properly-stored flour.

In the second place, when flour is made into bread, what change will contained particles of steel undergo? The following observations were carried out by mixing steel "cuttings" with flour, and making small loaves by means of German yeast, nothing further being added: "cuttings" were selected in preference to filings, since their greater size allowed of their more ready study. The cuttings were washed in ether and absolute alcohol, and dried immediately before use.

May 21, 1916: A loaf was made in the manner described. It was broken across twenty-four hours later. The particles of steel so exposed, and which are embedded in the paste forming the partitions of the vesicles, appeared of a dull black or dark grey colour. In order to isolate them from their thin investment of starch paste, some were picked out and boiled in a test tube in distilled water, being at the same time brushed against its bottom; they were then transferred to absolute alcohol in a Petri dish. A control observation upon unused steel cuttings showed that the boiling for this short period produced no change whatever. When viewed on a black ground they were all found to be dull black or grey, here and there blotched with pale red. Under the microscope when examined in Farrant's medium, by direct light, and on a dark ground, they exhibited scintillating points and brightly punctated lines (the marks of the tool), but they were blotched with black (suboxide of iron), and yellowish red (ferric oxide). One fragment of the loaf was allowed to dry: no change had occurred in the particles at the end of six days. Another fragment was kept in a moist chamber; at the end of the same period no further change had taken place, the cuttings remaining to the naked eye of a dull grey or black colour.

This observation was confirmed by a second. The oxidation is extremely superficial; the layer of yellowish red, translucent ferric oxide, indeed, is so thin as to allow of the glint of the subjacent metal appearing through it.

May 21, 1916: In the next observation the loaf was made with self-rising flour to which steel cuttings had been freely added. When broken across the following day the cuttings appeared of a dull greyish black colour. Some were picked out and boiled, &c., as already described. Viewed on a black ground in a Petri dish of absolute alcohol, they were all tarnished, and pale orange in colour, though some reflected light to the naked eye. On microscopic inspection in Farrant's medium, they exhibited the usual glinting surface, but this was blotched with translucent ferric oxide.

Finally, steel cuttings were mixed with the finest white flour, and made into a paste without yeast, and baked, the material being thus devoid of any vesiculation. Twenty-four hours later, the flat cake was broken across: the steel was not obviously rusted. Some of the particles were picked out, boiled and brushed in distilled water, and inspected in absolute alcohol on a black ground; they all presented a pale orange yellow tint, and in a microscopic preparation made in Farrant's medium, they exhibited the usual lightly punctated lines, but blotched with pale orange from superficial oxidation.

It appears from these observations, then, that the steel particles in flour, when the latter is made into loaves, undergo very superficial oxidation, and that their protection in the films of the paste which forms the walls of the vesicles secures them against further change.

Thirdly, what changes will take place in iron or steel, or in ferric oxide, after ingestion? And these have to be considered in the stomach, the small intestine, and the colon. The following observations, although carried out *in vitro*, may be taken as applicable to the question:—

(a) *In the Stomach.*—When iron or steel filings are thinly distributed in a Petri dish and covered with a solution of HCl of the percentage in which it is present in the human gastric secretion, and incubated at 37° C., an abundant evolution of H almost immediately proceeds, with the resulting production of soluble ferrous chloride. If the fluid is tested after two hours by means of ferricyanide of potassium (2 per cent.), it gives an intense colour reaction (Turnbull's green); with ferrocyanide of potassium (2 per cent.) freshly prepared, it gives a sky blue whitish cloud,—reactions of ferrous salts: the clear Prussian blue reaction with ferrocyanide of potassium, demonstrative of ferric salts, is quite wanting; with ferricyanide of potassium ferric salts yield a pale olive green. In the further statements regarding ferrous and ferric salts, these are the reactions which were had recourse to. The actual amount of solution, nevertheless, which would take place in the stomach within two hours (the period of digestion) is comparatively small, and particles of iron or steel would pass on with very little loss

of bulk, in the chyme, into the intestine, where their further history will be referred to subsequently. The fate of ferric oxide in the stomach is of interest, since this varies (*in vitro*) according as pure ferric oxide is used, or particles of steel or of iron, the surface only of which is rusted, and that only in parts; for in the case of steel ingested in bread, the particles would be in the condition last mentioned.

Observation 1.—A small amount of pure ferric oxide, in fine powder, was distributed over the bottom of a Petri dish, washed in absolute alcohol, and when almost dry, covered with 10 cc. of HCl of the physiological gastric percentage; and incubated at 37° C. After an hour portion of the fluid was tested; the ferric oxide had undergone no recognizable diminution in bulk. It gave with potassium ferrocyanide only a faint, clear, sky blue, showing that a minute amount only of ferric chloride had been produced. No ferrous salt was present. After a period of two hours the reaction was the same, the blue being no more intense; the solution remained acid.

Q In a further observation, after a period of twenty hours' incubation, the Prussian blue reaction obtained was of no striking depth. And in yet another, where the incubation was prolonged for three days, the colour, though fairly deep, was not intense; the fluid itself was colourless, and no recognizable loss of bulk had occurred in the ferric oxide.

The solubility of ferric oxide in HCl of the physiological percentage is thus extremely slight. By contrast, if a 25 per cent. solution of HCl is used, the fluid acquires a pale yellow colour, and gives a deep Prussian blue reaction after an hour's incubation. If now the observation is carried out as it would occur under natural conditions, the result is curiously different.

Observation 2.—Some of the lightly rusted steel filings isolated from the loaf in which they had been baked, were washed, and incubated in the HCl solution of physiological percentage. Examination at the end of the first hour showed the evolution of numerous minute bubbles of H proceeding from the surface of the steel. The fluid gave only the ferrous reaction, and no trace of ferric, proving that ferrous chloride was alone present. At the close of two hours the reaction was precisely the same.

The explanation of the foregoing difference is that any ferric chloride formed is reduced by the free H to ferrous. It must be observed, however, that the relative insolubility of ferric oxide in HCl of the gastric percentage does not apply to the hydrated form. If the latter is prepared by adding ammonia to ferric chloride solution, the resulting precipitate of hydrated ferric oxide, washed in distilled water, and treated (without being allowed to dry) in a Petri dish with the

acid, it rapidly dissolves: after an hour's incubation at 37° C., the resulting pale reddish brown clear solution gives an intense ferric reaction with ferrocyanide of potassium, and no ferrous reaction with the ferricyanide.

Desultory as all this may appear, there emerges the important fact that rusting particles of steel ingested in bread would yield, not ferric but ferrous chloride, and the point of this is that any resulting intestinal stasis would be negligible. The astringent action of ferric chloride, on the contrary, is well recognized, although the excess of HCl in the pharmacopœal liq. ferri perchloridi intensifies it.

Let it be supposed, in the next place, that particles of ingested steel escape complete solution in the stomach, what will happen to them in the small intestine? And, as a matter of fact, the solution of iron (or steel) filings, *in vitro*, is by no means rapid, and would certainly be uncompleted under the natural conditions of gastric digestion, as may be deduced from the following observation:—

July 27, 1915: 20 mg. of iron filings were distributed over the bottom of a Petri dish, covered with 40 c.c. of HCl solution of the physiological, gastric percentage, and incubated at 37° C., the filings being shifted from time to time with a glass rod. Although the free evolution of H showed that solution of the iron was in progress, there was no obvious diminution in the volume of the filings at the end of three hours, or of seven. After twenty-four hours a fair amount still remained. The solution of HCl was now renewed; a few fine particles were present after another two hours: by the next day solution was complete.

On a gross scale, the solvent action of the gastric juice upon steel is witnessed in the case of foreign bodies which have been purposely or unintentionally swallowed. Here, however, the action extends over long periods. Two specimens illustrative of this are contained in the Museum of the Royal College of Surgeons.

(No. 2255'1, Roy. Coll. Surg.) The remains of a table-knife which was swallowed two months before death. The handle, which was of bone, has been completely dissolved; the rivets have fallen out, and about a third of the edges, and the point of the blade are destroyed. After death the foreign body was found lying in the greater curvature of the stomach.

(No. 2307'1, Roy. Coll. Surg.) The blades and other steel portions of three or more clasp knives which were swallowed by a soldier, and voided *per rectum*. The surfaces and edges of some are deeply corroded. The man recovered.

(b) *In the Intestine.*—The intestinal contents below the entrance of the ductus communis, it will be remembered, are alkaline, the alkalinity being due to the presence of sodium bicarbonate in the pancreatic secretion. The amount of this secretion in the human subject may be judged of from cases of pancreatic fistula. Sir John Bland-Sutton¹ records a case in which a duodenal fistula formed after a posterior gastro-jejunostomy carried out in conjunction with excision of the pylorus for carcinoma, the divided ends of the duodenum and of the stomach being closed. A few days afterwards the duodenal stump leaked, and bile-stained fluid escaped through the lower angle of the abdominal incision: there were times when the fluid, which was quite clear and alkaline, flowed in an almost continuous stream—this when the patient took milk, the great stimulant of the gland being ingested fat.

The action of sodium carbonate in preventing the oxidation of steel is well known, and is turned to practical account by adding it to the water in which surgical instruments are boiled for sterilization. Upon iron filings observations were made with sodium carbonate (Na_2CO_3) and with acid sodium carbonate (NaHCO_3 ; sodium hydrogen carbonate, or sodium bicarbonate). The solutions were made in distilled water (first boiled and cooled), and of the percentage in which the salt is present in the pancreatic secretion. In the former, iron filings may be kept indefinitely without undergoing oxidation.

July 21, 1915: Iron filings were thinly distributed over the bottom of a Petri dish, covered with a freshly made solution of sodium carbonate, and incubated at 37°C . No oxidation had occurred at the end of fourteen days. In another observation, made in a wide test-tube, closed with a rubber cork, the filings were incubated for eight weeks: at the end of that time the fluid was quite clear, and the particles of iron were absolutely unaltered.

If acid sodium carbonate (sodium bicarbonate) is used, a very limited formation of ferric oxide results.

September 16, 1915: An experiment similar to the foregoing was made with the bicarbonate, the distilled water being boiled and cooled before the addition of the salt. (If the solution is boiled after its preparation, CO_2 is disengaged and the bicarbonate reduced to carbonate.) In twenty-four hours the particles of iron adhering to the sides of the lower part of the tube, all presented a yellow halo of ferric oxide, secondarily produced by the oxidation of ferrous carbonate formed from the combination of the second molecule of CO_2 with the iron. The change did not progress, and even on October 4, the oxidation had not obviously extended, its continuance being prevented by the solution having been now reduced to one of sodium carbonate.

¹ *Med. Soc. Trans.*, 1915, xxxviii, p. 2.

For a similar reason in water naturally containing carbon dioxide, rusting rapidly occurs from the formation of ferrous carbonate which readily undergoes conversion into ferric oxide—a transformation which takes place whether ferrous carbonate is in a precipitated form, or in solution, as it is in certain natural waters. That oxidation of iron may take place in water in the complete absence of dissolved oxygen is an observation of which the explanation is, that a mere want of structural homogeneity in iron suffices to set up electrolytic changes in the surrounding medium which bring about its decomposition. The property of sodium carbonate in preventing oxidation depends upon



FIG. 5.

Iron oxides obtained by allowing pure iron wire to rust in distilled water. On the right-hand side are shown translucent, orange-red or yellow, short bacilloid forms, as well as groups of larger crystals, both consisting of ferric oxide. On the left are similar forms which are black and opaque; these consist of suboxide of iron, and undergo subsequent conversion into proper rust or ferric oxide. Examined in Farrant's medium. ($\frac{1}{4}$ obj.)

Ferri suboxidum (in parte sinistra) et ferri oxidum (in parte dextra) in glycerino examinata. Utriusque, et figurae bacilloides et crystalli, inter se simillima sunt. Ferri oxidi, autem, particulae, translucentae atque rubrae.

its inhibiting the action of the hydrogen ions present in water. This inhibition is so effective that even if a solution of sodium carbonate is daily aerated by shaking, no rusting of iron filings submerged in it will

take place. In contrast with this, if iron filings are distributed over the bottom of a Petri dish, covered with distilled water, and incubated at 37° C., a striking amount of rusting occurs within twenty-four hours. This is equally true whether the distilled water has been first boiled to drive off dissolved air or not.

The following figures of ferric oxide from different sources are appended in order to facilitate their microscopic recognition. The first of them (fig. 5) is from short lengths of pure iron wire, washed in ether and absolute alcohol, and allowed to rust in a Petri dish at 37° C., in distilled water which had been boiled and allowed to cool. Within



FIG. 6.

Ferric oxide (from the Chemical Laboratory) examined in Farrant's medium ; translucent spherules of orange colour and varying size. ($\frac{1}{2}$ obj.)

Ferri oxidum in glycerino examinatum ; sphaerulae translucidae et rubrae, in magnitudine diversae.

twenty-four hours each piece was coated with a deep black layer of suboxide, most of the surface of which had been converted into finely-divided, orange yellow ferric oxide. On swirling the dish, much of the double investment was removed ; a microscopic examination of it, in Farrant's medium, showed flakes or collections of smaller, translucent pale orange, short bacilloid forms, and groups of longer crystals, of ferric oxide. And besides these, similar forms of both kinds, of a black,

or greenish-black colour, consisting of suboxide of iron. The red forms are derived from the black (fig. 5). Microscopic inspection of the wire so washed, showed incomplete groups of parallel lines of scintillating points, with unreflecting black areas between; the glinting surface is in spots of a pale orange colour—i.e., the light is reflected from below through a thin investment of ferric oxide. On the bottom of the Petri dish each piece of wire left a double line of black (suboxide) with clear intervening space, and a halo of red (ferric oxide) on the outer side; both of these were readily dissolved in HCl. Observations made by substituting steel filings for the iron wire, gave precisely similar microscopic results on examining the swirled-off oxides in Farrant's medium.

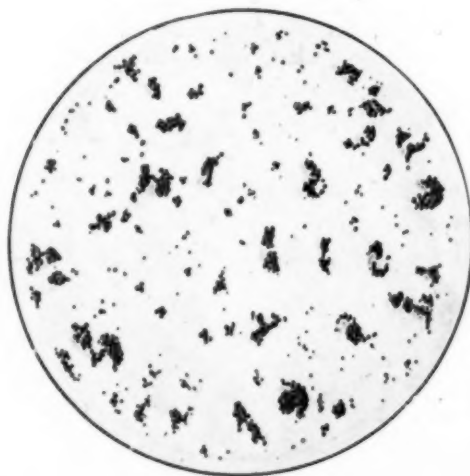


FIG. 7.

Finely ground native ferric oxide, as used for oil paint; examined in Farrant's medium. ($\frac{1}{4}$ obj.)

Ferri oxidum minute pro pigmento tritum; in glycerino examinatum.

Fig. 6 shows the ferric oxide used in the chemical laboratory. It consists of short, bacilloid, translucent forms of beautiful orange colour, and varying slightly in size.

The ferric oxide used for oil paint is shown in fig. 7; it consists of deep red, opaque particles, finer than the preceding. The material is found native, and is finely ground for the purpose required.

The most delicate microscopic form of ferric oxide is the hydrated.

The specimen from which the accompanying figure (fig. 8) is taken was obtained by adding ferric chloride to ammonia solution and washing the resulting loose, reddish-brown precipitate of hydrated oxide with distilled water. The examination of the precipitate was made in Farrant's medium, without its having been allowed to dry. It consists of soft-looking, pale yellow, transparent flakes, having a finely granular structure, much like colonies of micrococci.

The intestinal contents, like the gastric, contain a certain amount of free oxygen derived from air ingested during deglutition; but one of the observations referred to indicates that this would be inoperative to produce oxidation in the presence of sodium carbonate.



FIG. 8.

Hydrated ferric oxide. Collections of fine granules much resembling bacterial colonies. ($\frac{1}{4}$ obj.)

Ferri oxidum hydratum; *particulæ minutæ ut in micrococcorum coloniis aggregatæ*. In glycerino examinatum.

The conclusion, therefore, is that particles of steel, after ingestion in bread, would traverse the stomach and small intestine without undergoing chemical change, except for the small amount which would be converted into ferrous chloride in the stomach by the action of the

free HCl of the gastric juice; and that any encrusting ferric oxide which was undissolved by the same acid, would remain unchanged in the small intestine. Ferric oxide may be incubated in a solution of sodium carbonate or bicarbonate, of the pancreatic percentage, indefinitely, without undergoing any change whatever. The only reservation to this statement is that the action of free CO_2 in the small intestine might produce a minute amount of ferrous carbonate which would subsequently become converted into ferric oxide; for CO_2 will be liberated during the neutralization of the sodium bicarbonate of the pancreatic juice by the HCl of the chyme passed on from the stomach; and it is, moreover, liberated in the bacterial fermentation of sugar in the small intestine. Any ferric oxide so produced would be incapable of further change in the alkaline contents of the small intestine. The conversion of ferrous carbonate into ferric oxide is one that occurs very readily and rapidly in solutions, and it is not inhibited in an excess of sodium carbonate, when formed—e.g., by adding ferric chloride to a solution of the last-named salt. Particles of steel ingested in bread must, consequently, reach the large intestine, either in a metallic form or thinly invested with ferric oxide.

Finally, in the large intestine, both metallic iron and ferric oxide would become converted wholly or superficially into ferrous sulphide by the action of the hydric sulphide produced by the agency of the colon bacillus. This may be readily observed, *in vitro*, by passing hydric sulphide through distilled water in which either iron filings or ferric oxide are distributed. When filings so treated are examined microscopically they are found to have entirely lost their brilliancy, and to have become absolutely black from the superficial formation of ferrous sulphide. Ferric oxide, if similarly treated, rapidly blackens for the same reason; if it is afterwards washed in distilled water, it reacquires the characters of ferric oxide, the more readily as the particles are only superficially altered; both changes occur particularly rapidly in the case of the hydrated oxide. Ferrous sulphide, even when prepared by precipitation, will, when washed and left in distilled water, undergo conversion into yellowish brown ferric oxide; in microscopic preparations, the latter, under such circumstances, consists of very fine yellowish granules, corresponding in size with those of the ferrous sulphide. The production of sulphide results in the familiar blackening of the faeces which follows the administration of iron.

Pigmentation of the Appendicular Mucosa.

The pigmentation of the mucosa, which was discovered by Mr. W. H. Battle in a certain number of appendices (six in all) he has excised for disease, has been adduced by him in support of the hypothesis that the long continued ingestion of steel in bread is an important cause of appendicitis. And it seems at first as though the occurrence contributed evidence in this direction since there was no history forthcoming of the patients having taken iron medicinally. Of four of these specimens I have made a careful microscopic and microchemical examination: one of these is in the Museum of the Royal College of Surgeons, and three (1096B, C, D), in that of St. Thomas's Hospital. The histological findings are so alike that one description will suffice for all. The microscopic sections show that the pigment lies in the tissue surrounding the crypts, so as to be roughly disposed in vertical but ill-defined or broken lines; there is none in the muscular coat. It is contained in



FIG. 9.

One of the pigmented appendices (No. 1096D, St. Thomas's) referred to. The pigmentation is less intense towards the distal end. The small uncoloured circular foci correspond with the lymphatic nodules, which are devoid of pigment. From a woman who had suffered from chronic intestinal stasis, and sickness, with pain in the right iliac region for some months. (Natural size.)

Appendicis nigrities. Tela lymphoidea interglandularis membranae mucosae solum tingitur. A nodulis lymphaticis pigmentum abest. (Magnitudinis naturalis.)

clusters of large polymorphous cells which lie in the peritubular lymphatic tissue. Here and there a similar cell is to be encountered in the submucosa. There is no pigment in the lymph nodules; and as these (like the solitary glands of the intestine) lie in, and not beneath, the mucosa, they stand out to the naked eye as small white circular foci on the dark background formed by the rest of the surface (fig. 9). In unstained microscopic sections the pigment consists of granules, associated with larger spherules, of a dull brown or yellow brown colour. When sections are freely treated in the open with pure HCl

no solution of the granules, or discharge of their colour takes place. If the acid is afterwards diluted on the slide no ferric nor ferrous reaction is obtainable.

In carrying out microchemical tests for iron in sections, the use of heat is an important adjunct, and will sometimes bring about a reaction not obtainable in the cold. The method adopted was one arrived at by Dr. A. D. Gardner a few years ago at St. Thomas's Hospital, although the method of heating had been previously discovered and its importance insisted upon by Miss Maude E. Abbott.¹ The section, whether



FIG. 10.

Six connective tissue cells selected from the lymphoid tissue between the crypts in one of the pigmented appendices referred to. They are in varying degrees filled with granules and coarser spherules of a dull brown colour. The structure of the mucosa was otherwise normal. (2 obj.)

Appendicis nigrities. Cellulae quaedam selectae e telâ lymphoideâ membranae mucosae appendicis nigrescentis. In gradibus diversis cum moleculis et sphaerulis fuscis implentur. In structurâ microscopica membrana aliter normalis. Pigmentum nullas ferri notas microscopicas monstrat. Non dubitari potest quin ex sanguine aut in circuitu (haemochromatosis) aut ab appendicis lumine absorpto ortum sit.

paraffined or frozen, is treated on the slide with the following mixture for ferric reaction: 2 per cent. freshly made potassium ferrocyanide,

¹ "Pigmentation Cirrhosis of the Liver," *Path. Soc. Trans.*, 1900, li, p. 66.

two parts; 25 per cent. HCl, one part. The slide is now warmed till it steams, for two minutes or more, when the fluid is poured off and the section mounted in Farrant's medium, or in balsam after the usual procedures. A similar method may be adopted for testing ferrous salts, substituting 2 per cent. recently made ferricyanide for ferrocyanide of potassium. Both tests were systematically applied in the case of all four of the pigmented appendices; the use of steel instruments being, of course, carefully avoided throughout the manipulations. In not one did the pigmented granules or spherules undergo solution or show any blue coloration, either with the ferro- or the ferri-cyanide solutions. These results prove conclusively that the pigment does not consist of any form of free or inorganic iron; and, moreover, that no organic iron admitted of being unmasked in it.¹

As previously pointed out, iron carried from the lumen of the appendix into its wall can only be (1) in the metallic state, or (2) as ferric oxide, or (3) ferrous sulphide; and any of these would be soluble in HCl. Furthermore, the microscopic forms of all these have already been figured, and none resemble, in colour or form, the spherules present in the pigmented appendices. And, finally, one of the pigmented appendices—viz., that shown in fig. 9—gave a picture of no greater depth than a normal appendix when submitted to the Röntgen rays.

There is, however, in the collection of the Royal College of Surgeons a remarkable specimen of Hunter's, in which a large amount of ferric oxide was taken after death from an adventitious sac around a perforated appendix, and which may be here worth citing. (No. 2278'1.)

The ferric oxide occurs in large, friable, light, amorphous masses of brick red colour. A close inspection of the fractured surfaces reveals the presence of a considerable number of somewhat fine animal hairs. Microscopic preparations show the presence, moreover, of fragments of undigested vegetable tissue, but the most conspicuous element consists of large numbers of minute, translucent particles of orange yellow colour, which consist of ferric oxide; fat, calcium soaps, and a little phosphate of lime were also discovered by chemical examination. The source of the ferric oxide is not given in the history of the case, which is taken from the Hunterian MS. It was probably derived from iron taken in some form medicinally (fig. 11).

The material on solution in HCl gives a ferric reaction; and it

¹ Three of the appendices were fixed in formol; one, in alcohol only.

blackens from the formation of ferrous sulphide, when H_2S is run through a suspension of it in distilled water. The fine state of its division, as seen under the microscope in Farrant's medium, suggests that the ferric oxide has been in the hydrated form—i.e., it is not ferric oxide ingested as such, but probably derived from ferrous carbonate, or some other form of iron, taken medicinally: from the stomach any ferrous chloride therein produced, which passed into the intestine would, on reaching the sodium carbonate of the pancreatic secretion become ferrous carbonate; this would readily undergo conversion into hydrated ferric oxide, after which it would be insusceptible of further change,

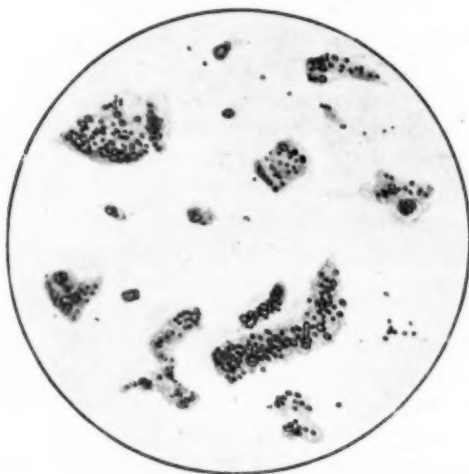


FIG. 11.

Ferric oxide, examined in Farrant's medium, from a sac in connexion with a perforated appendix (Hunter's case). Its fine state of division suggests that it was of the hydrated variety; it was probably derived from iron taken in some form medicinally. ($\frac{1}{4}$ obj.)

Ferri oxidum e cavitare cum appendice communicanti extractum. In formâ quâdam ferrum forsitan datum erat.

except that in the colon it might become converted into ferrous sulphide. The reconversion of the sulphide into ferric oxide on exposure to the air occurs as readily as does the conversion of the oxide into sulphide under the action of hydric sulphide.

In the case of the pigmented appendices, it is easier to say what the pigment is not, than what it is.¹ In their colour, general form, and intracellular location, the granules and coarser spherules at once recall those of blood pigment. The absence of iron on this supposition offers no difficulty. For the pigment derived from effused blood is, of course, at times iron-free (hæmatoidin), whilst at others it gives an intense blue, ferric reaction (hæmosiderin); and both forms may be present together. Further than that, one may encounter pigments which give both a ferrous and a ferric reaction.

This was so in the microscopic sections cut from an organized thrombus or "polypus" attached to one of the curtains of the mitral valve (Royal College of Surgeons Museum) of a woman who died suddenly; doubtless from heart failure. The pigment, which is intracellular, is spherular in form, and of a blackish brown colour; with ferrocyanide of potassium and HCl it became intensely blue (Prussian blue), and with ferricyanide and HCl it likewise gave a well pronounced greenward blue (Turnbull's blue). Whether the same spherules gave both reactions—i.e., whether they contained a ferroso-ferric compound like the magnetic oxide found native ($\text{Fe}_3\text{O}_4 = \text{Fe}_2\text{O}_3 + \text{FeO}$), or whether the two reactions were given by different groups of spherules, may be questioned; but I believe that the first was the case, since the groups were as large individually, and appeared about as numerous in both preparations. This ferroso-ferric reaction was very conspicuous in the cases of pigmentary hepatic cirrhosis to be presently mentioned, where it was evidently given by the same granules.

The pigment in the appendices referred to is derived, not from blood extravasated into the tissue, but from blood either absorbed from the lumen, or from that in circulation (hæmochromatosis). Of the 125 normal appendices which I have examined, blood was present in a certain number, chiefly in cases of melæna due to gastric or duodenal hæmorrhage. Blood may be extravasated, again, from ulcers in the appendix itself. I have, however, only once seen blood within the lumen of an excised appendix—in a case operated upon by Sir Rickman Godlee.

That a certain form of pigmentation may result from the interstitial effusion of blood into the tissue in an acute hæmorrhagic appendicitis must not be lost sight of. In the Museum of St. Thomas's there is an excised appendix (No. 1107 N), the mucosa of which is

¹ As regards some, inquiry showed that the patient had not medicinally taken iron, or bismuth, or mercury.

irregularly blackened from hæmorrhagic inflammation, where dilated around a group of concretions. Microscopic examination shows that the mucosa has been extensively destroyed, the deeper portions of the crypts alone persisting; and that only in places. Besides the presence of free blood in the remains of the mucous membrane, large numbers of deep brownish black granules are distributed singly and in clusters in the lymphatic tissue between the remnants of the glands, as well as in the epithelium of the latter; some of the pigment is in polymorphs, of which an abundant migration has taken place. In sections heated in ferrocyanide or ferricyanide of potassium and hydrochloric acid the granules remain quite unchanged—i.e., they are iron free; as they do, also, under the action of pure HCl. The picture presented under these circumstances is altogether different, however, from that of the appendices first described, where the discoloration is remarkably uniform, and the mucosa, where pigmented, may be quite intact and covered with a normal epithelium. An inflammatory source of the pigmentation in these may thus be excluded.

Has the pigment in the different appendices removed by Mr. Battle been deposited from the circulating blood?

In cases of hæmochromatosis, blood pigment so deposited retains its iron, and gives the usual ferric reaction; this is not so in the appendices referred to. Not only is this true in pernicious anæmia, but it is so in the examples of hepatic cirrhosis that have been named "pigmentary."¹ In quite a limited number (five) of examinations of typically hobnailed organs I found three of the pigmentary kind. Kretz² discovered the hæmosiderin pigmentation in fourteen of twenty-six, and Miss Abbott in six of sixteen cirrhotic livers. In the first of those examined by myself, the macroscopic coloration was striking; the organ being externally of a nut brown; and, in section, regularly mottled, from the colour being most intense in the perilobular connective tissue, the lesser depth in the nodules of hepatic substance being due to distension of the hepatic cells with fat. When treated, after fixation in formol, with ferrocyanide of potassium and HCl, a blue as intense as any yielded in pernicious anæmia rapidly developed; slices immersed in ammonium sulphide became of a deep black colour, the connective tissue more particularly so. The histological study of

¹ A concise notice of this subject is given by Miss Maude Abbott, *Trans. Path. Soc.*, 1900, li, p. 66.

² Kretz, "Hæmosiderin Pigmentierung der Leber und Leber-Cirrhose," *Beitr. z. klin. Med. u. Chir.*, Wien, 1896, Heft 15.

unstained sections in Farrant's medium revealed the presence of vast numbers of yellowish or orange brown granules in the hepatic cells (many of which are distended with droplets of fat, around which the granules lie), and in the epithelium of the bile ducts; whilst free, and in the connective tissue cells of the interlobular fibrous tissue, the pigment was still more abundant and chiefly as massive collections of coarse spherules. The pigment is everywhere of the same colour. When sections are heated in potassium ferrocyanide and HCl, the granules and spherules, in the different positions mentioned, stain of a deep blue (Prussian), the whole section becoming to the naked eye of a beautiful colour. In sections heated with potassium ferricyanide and HCl, the coloration is as general and almost as deep a blue, with the faintest approach to green (Turnbull's blue)—i.e., the granules give both a ferric and a ferrous reaction.

With a view of testing the possibility of diagnosing such an iron-laden condition during life I have had a skiagram taken of a slice of this liver, and another of a slice of exactly the same thickness, from a normal liver similarly hardened in formol, for the purpose of comparison. An inspection of the accompanying figures will show that there is no increase of opacity in the pigmented organ, and that skiagraphy would be useless as a means of clinical diagnosis.

The other two examples of pigmentary cirrhosis, I found in a collection of four typically hobnailed organs, which are at present stored in the pathological collection of teaching specimens. To cite only one, for they are practically indetical: The organ was of a reddish brown colour, the pigmentation affecting the lobules, and only to a small degree the interlobular connective tissue. The pigment in microscopic sections, in both the positions named, gives an equally well marked ferric and ferrous reaction. In a fourth case the liver was of a dull reddish brown colour, smooth externally; the cirrhosis, monolobular. All the hepatic cells held large numbers of deep brown granules, and the latter were present, also, in the epithelium of the bile ducts, and in some of the connective tissue cells of the interlobular connective tissue. The granules in these different positions gave a deep ferric (Prussian blue) reaction, and an equally pronounced ferrous (Turnbull's blue). The spleen, which was slightly enlarged, likewise gave an iron reaction; lungs, a little congested; kidneys, normal to the eye. The patient, addicted to alcohol, died from encephalitis of one of the occipital lobes. The blood was not examined; nor was the urine, except by the routine methods. It may be stated that this liver, like the others, had been hardened in formol solution.



FIG. 12.

A skiagram of a slice, 1.7 cm. ($\frac{5}{8}$ in.) in thickness, from the liver of a well-nourished man who died of acute food poisoning, hardened in formol. The normality was corroborated by microscopic examination; the hepatic cells in the peripheral zone of the lobules, were distended with fat (Scharlach stain), but within the physiological limit. Some of the cells in general held a few fine granules of pigment. (Natural size.)

Pictura radiographica. Hepatis normalis portio in crassitudine 1.7 cm.; in liquore formaldehydi condita. (Magnitudinis naturalis.)



FIG. 13.

A skiagram of a slice of liver of exactly the same thickness as the preceding—viz., 1·7 cm. ($\frac{5}{8}$ in.), from a highly pronounced case of pigmentary cirrhosis. The organ was hobnailed on the surface, and gave an intense blue colour when treated with ferrocyanide of potassium and hydrochloric acid. Although the hepatic cells are laden with granules of hæmosiderin the rays have passed through as readily as in the case of the normal tissue. (Natural size.)

Hepatis cirrhosis pigmentosa. Pictura radiographica. Hepatis portio in crassitudine 1·7 cm., in liquore formaldehydi condita. Etsi cellulae hepaticae hæmosiderini granulis impletæ sunt, radii tam facile percolantur quam in hepate normali præcedenti. (Magnitudinis naturalis.)

Excluding the cases in which hepatic, and more generalized, hæmosiderosis is associated with pernicious anæmia, chronic pancreatitis hereditary jaundice, &c., it is clear that alcoholic cirrhosis is in certain cases complicated by some other factor, which needs investigation. Unfortunately a skiagram would not disclose the pigmentation in life, and to strike a case for special investigation would involve a systematic examination of the blood and urine in every patient suffering with hepatic cirrhosis. The bile pigment, though derived from the hæmoglobin, does not, as is well known, contain iron. And the alternative hypotheses in pigmentary hepatic cirrhosis are: (1) that the blood is hæmolysed before its intussusception into the hepatic cells; and (2) that the hepatic cells, owing to damage from disease, are incapable of removing the iron from the bile pigment. There are, it must be confessed, no histological differences between hobnailed livers which give an iron reaction, and those which do not. And the deposit of hæmosiderin is more probably the result of hæmolysis complicating the other disease.

To return, in conclusion, to the appendicular pigmentation; whether it is due to the absorption of blood from the lumen, or from blood in circulation, cannot be with certainty decided, since the condition of the large intestine is unknown: it might be either, for in its characters the pigmentation is identical with that in the cæcum to be next described, where it is so uniform and general that its hæmochromatic nature cannot be doubted. The absence of iron from the pigment in these specimens does not exclude the latter mode of origin; for the iron may have been removed from the pigment after the deposition of this from the blood, as it may be after its deposition from a local extravasation.

To the appendices, then, may be added the following specimen of cæcum from the College Museum, since the pigmentation is not of the kind due to any form of metallic deposit: A vermiform appendix bisected, with the adjacent portion of the cæcum; the appendix, of which the walls are slightly thickened, is occluded at its orifice, and near its middle is subdivided by a horizontal diaphragm, as results of past inflammation. The mucosa of the cæcum is of a deep reddish-brown colour, the pigmentation ceasing abruptly at the spot where the lumen of the appendix is closed. Microscopic examination shows the coloration to be due to the presence of spherular pigment contained mostly in cells between the crypts of the mucosa. The histological details in this case are identical, in short, with those of the appendices: the micro-chemical tests for iron being quite negative; nor was the spherular

pigment affected in sections when freely exposed to the action of strong HCl and warmed. A considerable piece of the pigmented mucosa was boiled in pure HCl, the fluid filtered, diluted with distilled water, and tested for iron: no reaction was obtained with ferrocyanide of potassium, nor any red coloration with potassium sulphocyanide; the Turnbull green, or ferrous, reaction with potassium ferricyanide was likewise negative. The parts were removed from a woman, aged 58, previously healthy, who had an attack of diarrhoea on October 28, 1914, and got thoroughly chilled on the following day. On October 30, she complained of severe abdominal pain; the temperature was 99° F. to 100° F. On October 31, there was excessive tenderness on deep pressure over the region of the appendix, which was removed the same day: it was bound down by adhesions. Nothing relative to the cause of the pigmentation was elicited. One point of interest in this specimen is the absence of pigmentation in the appendix. At first this seems sufficient to prove that the pigmentation is not derived from the circulating blood, since were it otherwise, seeing that the local vascular distribution is not deranged, the deposit would have occurred in the appendicular mucosa as well. A microscopic examination, however, shows that the mucosa has been completely destroyed as a result of some past process. The muscular coat is normal; the muscularis mucosæ easily recognizable; but the mucosa is represented only by a well-defined layer of connective tissue in which are a certain number of eosinophile cells; there are no traces of crypts, and the scar tissue has no investing epithelium. As there is no mucosa present, the specimen does not dispose of the view that the pigment may have been deposited as an *internal excretion* from the circulating blood—a term I venture to suggest for such depositions of effete autogenous material in the connective tissues, unattended with actual extrusion from the body.

And somewhat similarly, in one of the pigmented appendices previously referred to (St. Thomas's, 1096C), the terminal portion for a distance of 4 cm. (1½ in.) is contracted and obliterated, and is here quite devoid of colour. This specimen was excised from a woman, aged 48, who had suffered from several attacks of pain and inflammation in the region of the appendix. She had taken no medicine containing iron.

The possibility of the intramucosal pigment being furnished directly from vegetable ingesta must not be passed by. The deeply coloured bezoars of Ellagic acid from the stomach of the goat, ape, &c., have

a purely vegetable origin. Externally these enteroliths are remarkably smooth and polished, and are of so deep a brown as to look almost black. In section they are concentrically laminated, and they usually display a seed or a twig in the centre, in which latter case the concretion is sometimes curiously elongated or pencil shaped. No such calculus has been hitherto observed in the human subject. The material, on microscopic examination, presents a fine concentric striation, in addition, i.e., to the coarser which is obvious to the eye; the lamellæ themselves are of crystalline structure, the elements being set in general with a radial disposition. When the material is finely ground and examined in glycerine, it is seen to consist of minute, multiform, angular particles, a few spheroidal, passing into coarser fragments which are concentrically and radially striated: none are of more than the palest yellow; and neither in shape nor colour do they



FIG. 14.

A skiagram of half of a bisected bezoar (or enterolith from one of the lower mammals) consisting of ellagic acid. The substance has allowed a free passage to the rays. The light central area represents a cavity from which an elongated nucleus has been removed. (Roy. Coll. Surg., Q2.) (Natural size.)

Enterolithus acidi ellagici (bezoar ut dicitur) capri ex ventriculo forsitan extractus. Monstrat pictura radiographica radios per concretionem facile percolari. (Magnitudinis naturalis.)

resemble the pigment contained in the mucosa of the appendices that have been described. The ellagic acid of which they consist is soluble in cold caustic potash solution, an ellagate of potassium being formed from which the acid is recoverable by means of HCl as a finely divided, dirty buff precipitate; if examined microscopically this will be found to consist of granules of the same pale yellow colour. When microscopic sections of the pigmented appendices, and of the pigmented cæcum, already described, are freely treated in the open with caustic potash

solution, the pigment granules, like the rest of the tissue, swell, but they are not dissolved, nor is the colour discharged. In this, again, they behave like blood pigment.

With regard to the bezoars of Resino-bezoardic acid, which burn freely with a smoky flame, and have likewise a vegetable source, it is enough to say that they are practically without colour, and that no such enteroliths have been observed in the human subject. When submitted to the X-rays they exhibit, I find, a medium degree of opacity, about the same as that, e.g., of a calculus of cystine. To the naked eye the section is finely and regularly laminated, and when crushed and examined in Farrant's medium, the substance appears as transparent, crystalline, and almost colourless, disposed in fine concentric lamellæ, which have a radially crystalline construction. The material is readily soluble in caustic potash, and in boiling alcohol.

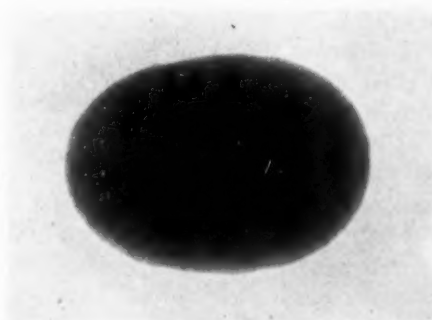


FIG. 15.

A skiagram of half of a bisected enterolith of resino-bezoardic acid, from one of the lower animals. The substance gives a deeper shadow than the ellagic acid enterolith. (Roy. Coll. Surg., R7.) (Natural size.)

Enterolithus acidi resino-bezoardici ex animali quodam herbivoro. Pictura radiographica. (Magnitudinis naturalis.)

The familiar colour of bran is due to the presence of a pale brown homogeneous material in certain of the subepidermal cells of the pericarp. The material is insoluble in alcohol or caustic potash, but cannot be related in any way to the pigmentation of the appendix.

Although it is sufficient for the present purpose to exclude the presence of inorganic iron in regard to pigmentation of the appendix,

certain metallic sulphides may be excluded as well. *Sulphide of mercury* can be excluded, amongst other ways, by the microscopic form and colour of the pigment. In the College Museum there is a piece of the colon from the oft-cited case of mercurial pigmentation described by Dr. C. J. Williams.¹ It is of a deep black colour, and came from a patient who had been in the habit of taking a grain of calomel (HgCl) every night for forty-three years. After death the whole of the large intestine was found of a deep black colour, the small gut and stomach being quite free of pigmentation; the appendix was not examined. The presence of mercury was demonstrated in the wall of the colon by sublimation. The liver was cirrhotic, but no mercury was demonstrable chemically in it. Clinically, the case was remarkable owing to the fact that no salivation was induced, and no soreness of the gums or bone necrosis. That the metal is in the form of sulphide can be shown by boiling a portion of the detached mucosa in nitric acid; the tissue dissolves, but a number of fine dark particles remain which, when washed and examined microscopically, are identical with those present in the microscopic sections. Sulphide of mercury is the only metallic sulphide not soluble in nitric acid. Of this specimen I have, also, made a new series of microscopic sections, with the following result: The deposit consists of fine, translucent, chocolate brown granules, which when aggregated, appear of a deep black colour; it is confined to the mucous membrane and submucosa. The material is mostly free, but some is in well defined clusters as though within cells; discrete particles are freely distributed in the connective tissue bounding the crypts. In the submucosa the accumulation is much more extensive and occurs in long, deep black lines, the size and arrangement of which indicate that they are lymph channels. That this is so, is further shown by the circumstance that the contents have escaped from some of the clefts and only adhere to the inner sides of the latter; the spaces here appear as distinct canals with ampullæ, are of considerable length, and run horizontally in the submucous tissue.

Calomel is quite insoluble in HCl . If incubated in a solution of sodium bicarbonate of the pancreatic percentage, it becomes, within a few hours, of a grey colour from the superficial formation of suboxide. On microscopic examination the coarse, translucent crystals of the calomel are found besprinkled with fine, coccoid, blackish granules, others of which are free. A similar result presumably occurs in the

¹ *Trans. Path. Soc.*, 1867, xviii, p. 111.

small intestine. Calomel, suspended in distilled water, readily blackens under the action of hydric sulphide. That the coloration of the wall of the colon is not due to the simple absorption of such is clear from the microscopic characters of the deposit in it, for calomel so blackened still retains its original form of coarse, variously sized, and irregular particles, the formation of sulphide being only superficial.

If suboxide of mercury is prepared by precipitation (solution of potash to solution of subnitrate of mercury), the greyish black deposit presents itself microscopically as fine granules which are individually slightly translucent. If treated with hydric sulphide the colour is intensified from the production of sulphide of mercury. That the



FIG. 16.

A section of the wall of the colon pigmented from the presence of sulphide of mercury. The granules of the sulphide are particularly abundant in the submucosa; some of the deposit lies within lymphatic vessels, of which the plexus is recognizable.

Coli nigrescentis sectio microscopica canales lymphaticos monstrans granulis hydrargyri sulphidi impletas.

deposit in the colon is sulphide and not suboxide is proved by the fact, as before stated, that if boiled in strong nitric acid so as to dissolve the whole of the organic matter, a very finely divided black deposit shortly settles; sulphide of mercury being quite insoluble in nitric acid, whilst the suboxide is soluble. Ammonia forms with suboxide of mercury a

dull blackish compound ($\text{Hg}_2\text{NH}_2\text{Cl}$) which is equally insoluble in nitric acid, though like mercury sulphide it is soluble in a mixture of nitric and hydrochloric (aqua regia).

It may be concluded from these data that the coloration of the colon is due to a deposition from the circulating blood; and that the mercury is deposited in the first place as black suboxide, the colour of which has been intensified by its subsequent conversion into sulphide, possibly after death, from the access of hydric sulphide produced in the intestine. The pigmentation would thus be of the same hæmatogenous source as that of the skin which arises from the administration of silver solutions; or of the gums in lead poisoning. This conclusion is also borne out by the absence of any pigmentation in the small intestine: for suboxide (produced from calomel by the action of the pancreatic sodium bicarbonate), if absorbed as such in this part of the canal, evidently does not accumulate, but reaches the blood; whilst in the case of the colon the deposition is of the nature of an incomplete or internal excretion.

To this may be added, in order to complete the matter, the microscopic characters of a series of metallic sulphides precipitated from various solutions by allowing hydric sulphide to pass through them—viz., iron, mercury, lead, bismuth, antimony. The sulphide precipitates were washed in distilled water, and examined in glycerine, with $\frac{1}{2}$ objective.

Ferrous Sulphide.—Minute, deep black granules, about the size of staphylococci; mostly in somewhat large clusters.

Mercury Sulphide.—Small, translucent elements of brownish black colour; spherical or oval, aggregated in clusters: not so opaque or so black as ferrous sulphide. Some of the particles are larger and spherular.

Lead Sulphide.—Fine coccoid granules, discrete and clustered, of intensely black colour.

Bismuth Sulphide.—Fine coccus-like granules; discrete and aggregated in clusters of a deep brownish black colour; not so intensely black as lead sulphide.

Antimony Sulphide.—Irregular masses of translucent, bright orange-yellow coccoid particles.

APPENDICULAR CONCRETIONS.

The mechanical or traumatic causation of appendicitis cannot be adequately tested without considering how far the presence of foreign bodies is responsible for the production of appendicular concretions, since in a considerable proportion of cases the inflammatory process is associated with the presence of the latter.

One of the most striking things in regard to calculi formed upon foreign bodies is the disparity which not infrequently obtains between their size and that of their nuclei. The most remarkable examples of this disproportion are furnished by the intestinal calculi of ammonio-magnesium phosphate which are not uncommonly met with in the intestine of the horse, and which may be of huge dimensions, whilst the nucleus is invariably insignificant; a nail (often horse-shoe), a fragment of steel, a piece of flint, a small pebble, or a button. Once started, in fact, the calculus itself comes to furnish a growing nucleus for its own increase.

The two following specimens in the College collection are typical of their kind:—

(No. 2352.1, Roy. Coll. Surg.) Half of a smooth, spherical calculus, 9 cm. ($3\frac{1}{2}$ in.) in diameter which was taken from the intestine of a horse. The divided surface is throughout of uniform and compact structure, finely and regularly laminated in the concentric direction. In the centre is shown a small, flattened, oval pebble 1.8 cm. by 1.3 cm. in its chief diameters, which forms its nucleus.

(No. 2351, Roy. Coll. Surg.) Half of a large cubical calculus 9 cm. ($3\frac{1}{2}$ in.) in diameter, probably from the intestine of a horse. As displayed in the section it has a regularly concentric lamination, but no obvious radial construction except in the immediate neighbourhood of the nucleus, which consists of a small quadrilateral piece of steel only 0.8 cm. ($\frac{3}{10}$ in.) in its longer diameter. Although the nucleus is, comparatively, so minute, the form of the calculus exactly reproduces that of the foreign body.

The largest example of such equine enteroliths in the College (S3) is no less than 23 cm. (9 in.) in diameter; yet its nucleus is quite a small piece of steel, apparently a nail. As seen in museums, the face of these calculi is of an opaque, bluish-white colour, a condition brought about by the loss of the water of crystallization from the ammonio-magnesium phosphate; the recently exposed surface is of a pale brown and semitranslucent. In the case of urinary calculi

formed upon foreign bodies the same disproportion is to be witnessed. The extreme instance of it in the College collection is a phosphatic calculus 7.5 cm. (3 in.) in chief diameter, which has for its nucleus a hairpin which was inserted into the bladder (No. 2365'1, Roy. Coll. Surg.).

Mutatis mutandis, in the case of appendicular concretions, it might be anticipated that the magnitude of the nuclei would be, at times, correspondingly reduced. A single hair will serve, even in the urinary bladder, as the nucleus of a calculus. There are in the College Museum



FIG. 17.

A skiagram of half of a bisected equine enterolith consisting of ammonio-magnesium phosphate. Like calcium phosphate, ammonio-magnesium phosphate filters off the rays. (Natural size.)

Enterolithus, bisectus, equi ex colo extractus. Demonstrat pictura radiographica radios per ammonio-magnesium phosphatem, ex qua calculus solum constat, paulum modo percolari. (Magnitudinis naturalis.)

four examples of this, which bear sufficient relation to the present subject to be worth mention, and with respect to which the descriptions in the new catalogue will be enough.

(No. 2358'1, Roy. Coll. Surg.) Four small, flattened curved calculi (apparently phosphatic) formed upon pubic hairs. The largest measures 1.5 cm. ($\frac{7}{12}$ in.) in length. From one a piece has been

removed to exhibit the hair in its centre. From a gentleman, aged 82. The presence of vesical calculi was diagnosed with the lithotrite, and some were crushed, the débris being withdrawn. The bladder was subsequently washed out and about twenty similar concretions removed. Others had been passed naturally *per urethram*. The relief was permanent. The pubic hairs were presumably transferred to the bladder in the eye or on the surface of a catheter, which had been for a long while used upon the patient during the day and at night, and often more by feeling than by sight.¹

(No. 2359'1, Roy. Coll. Surg.) A small, matted lock of hair, each filament of which is covered with a continuous or moniliform deposit of phosphate; together with many calculi, the elongated form of which indicates their origin upon detached hairs. From a well-nourished married lady aged 30, the mother of two children, who, after an operation for piles, complained of frequency of micturition and vesical irritation. The urine contained mucus and pus. After the removal of this lock of hair, which was impacted in the urethra, the patient passed single hairs on many occasions, some of which were encrusted with phosphate. A digital exploration of the bladder was subsequently made, when a pedunculated tumour was discovered projecting from the upper part of the organ, and which apparently protruded from an annular depression in its wall; this tumour was completely removed. It was of the size and shape of a "button mushroom"; was thickly covered with hair, and microscopically was composed of fibrous tissue invested with skin furnished with sebaceous glands. The teratoma is preserved in the museum of Guy's Hospital. The patient was in good health many years afterwards.²

(No. Ha28, Roy. Coll. Surg.) A large collection of pyriform phosphatic calculi, averaging 1.5 cm. in length, which was passed *per urethram* during the years 1872 to 1879. From the tapering end of some there projects portion of a pale hair. In May, 1879, a calculus of more considerable dimensions was extracted from the bladder through the urethra (Ha29). After this, until 1893, a certain number of hairs encrusted with moniliform depositions of phosphate were passed (Ha 30); from that date until 1896 no further had appeared. The patient was a female, aged 69; the first calculi were voided when she was 44. Her hair was dark brown, becoming slightly grey. The source of the

¹ T. Bryant, *Trans. Path. Soc.*, 1901, lii, p. 163.

² T. Bryant, "A Manual for the Practice of Surgery," 4th ed., 1884, ii, p. 78.

hairs in this case was probably similar to that in the preceding—viz., a piliferous teratoma: in favour of this supposition is the fact that the hairs passed are pale in colour, whilst that of the patient was dark brown.

(No. Ha18, Roy. Coll. Surg.) Several phosphatic calculi varying in size from that of a mustard-seed to that of a large pea; some are perforated by short fine hairs, the bulbs of which in every case protrude from the concretions, showing that the hairs grew into the bladder and became detached after the deposition had taken place upon them. They were passed by a nurse who for ten years had suffered from impaired health, pain in the loins extending to the left kidney, pain and difficulty in micturition, and frequent desire to micturate. The urine was alkaline, deposited a slimy mucus, and was sometimes mixed with blood. These symptoms followed shortly after a fall down stairs, by which her back was injured, although at the time, she experienced but little inconvenience from the accident. Ever since the fall, scarcely a week went in which she did not pass one or more of such calculi.¹ Here as in the preceding cases, the hairs were doubtless derived from a teratoma projecting into the bladder.

There is lastly in the College collection a single oval calculus formed in connexion with hairs, in a male:—

(No. Ha19, Roy. Coll. Surg.) A flattened oval calculus, 4·7 cm. in chief diameter, which was extracted from the bladder of a man, aged 30. It consists chemically of mixed phosphates. In the mid-substance of the divided surface there is a line of subdivision, the central moiety being pyriform; and in the narrow end of the latter there are embedded a certain number of dark hairs. The patient had suffered eight years previously from a large dermoid cyst between the bladder and rectum. The cyst had been opened, and a considerable quantity of fluid fat mixed with hairs escaped. From this he had quite recovered; but no evidence could be procured that a communication had at any time existed between the cyst and the bladder.²

The discharge of ligatures into the urinary bladder after surgical operations, may be followed by similar results, as is illustrated by the three following specimens in the College Museum:—

(No. 2361·1, Roy. Coll. Surg.) Four silk ligatures from the urinary bladder. Upon one there has formed a spherical phosphatic calculus the size of a small cherry. Two of the others have, in places, small

¹ Edward Parker, *Edin. Med. and Surg. Journ.*, lxii, p. 151.

² T. B. Curling, *Path. Soc. Trans.*, xx, p. 238.

encrustations of phosphatic material adherent to them. From a lady upon whom ovariectomy was performed on March 24, 1884. On April 1, the sutures of the abdominal wall had been removed; union was perfect except for the opening through which the ends of the ligatures placed upon the peduncle passed. Later on the ligatures were drawn out as far as possible, and cut off short. In a few days the opening closed, and the patient needed no further treatment. She made a good recovery, but in June of the following year she complained of great pain in the lower part of the abdomen, and her temperature was raised. There was a discharge of pus from the bladder, and the encrusted ligatures were subsequently removed from it, an abscess having apparently burst within the viscus, and carried the ligatures into it.¹

(No. 2360-1, Roy. Coll. Surg.) A small collection of calculi which have formed in connexion with silk ligatures, which escaped into the bladder after the removal of a fibromyoma of the uterus. The upper two smaller, pyriform concretions were passed during micturition. The larger calculus below (which measures 3.2 cm. (1¼ in.) in length) was broken in two during its removal through the urethra; portion of a ligature can be seen in its centre. From a married woman, aged 48. In November, 1904, a fibromyoma was enucleated from the right mesometrium; the uterus was amputated above the cervix. The tumour had caused dysuria and other symptoms due to pressure. Ten months after the operation, rigors occurred, and much inflammatory thickening was found around the cervix. The dysuria returned with partial incontinence. In October, 1906, two of the calculi, as above noticed, were passed naturally; the other, larger stone was detected and removed a month later, being broken in two to allow of its more ready extraction by the urethra, which had been previously gradually dilated by bougies to take a No. 17. The patient soon regained control over the bladder.²

(No. Ha21, Roy. Coll. Surg.) The fragments of a large phosphatic calculus which formed upon a silver suture that had escaped by ulceration into the bladder of a woman, after an unsuccessful operation for the union of a vesico-vaginal fistula.

And to take another region of the body, as exhibiting an analogous phenomenon, there is in the College collection:—

(No. 2234-1, Roy. Coll. Surg.) A slightly curved slender fish-bone, 1.3 cm. (1½ in.) in length which was removed from Wharton's duct.

¹ J. H. Thompson, *Lancet*, 1885, i, p. 1123.

² Presented by Alban Doran, Esq., 1908.

For half its length it is thinly encrusted with phosphate. From a man, aged 63, who had experienced discomfort for two or three days, in the floor of the mouth. The sublingual salivary gland was slightly enlarged and tender. On examination a foreign body was found protruding at the orifice of Wharton's duct, from which it was thereupon extracted.

To pass now to appendicular concretions. A careful examination of a series of such shows that in a certain number, some foreign body forms a nucleus; whilst in others, none such is demonstrable—i.e., nothing is to be differentiated from the material composing the rest of the concretion. It is to be remembered, further, that a foreign body may be present in the appendix without any accompanying concretion. Doubtless (as in the case of foreign bodies in the urinary bladder) the element of time comes in with regard to the fact last mentioned, which is itself illustrated by the following specimens from the College Museum, and by others in that of St. Thomas's Hospital; the presence of thread-worms and whip-worms being excluded from consideration.

Pin (No. 2285'1, Roy. Coll. Surg.).—A vermiform appendix which was removed by operation. In its distal end there is a pin, of which the surface is deeply corroded; the walls of the appendix around the foreign body are much swollen from inflammatory exudation.

Pin (No. 2284'1, Roy. Coll. Surg.).—A vermiform appendix in which there is lodged a pin. The foreign body lies with its head against the distal end. The part was removed after death from the body of a young man, who died of pulmonary gangrene. The foreign body had produced no symptoms during life.

Pin.—There is a third example in the Museum of St. Thomas's Hospital (No. 1098B), taken after death from a man, who died with a subdiaphragmatic abscess, and multiple abscesses in the liver. The head of the pin lies about $\frac{3}{4}$ in. from the free end of the appendix, the portion of which corresponding with the foreign body is somewhat swollen from inflammation. The symptoms commenced eight weeks before admission with pain localized in the right iliac fossa. Death took place two months after his admission.

The following series will serve as further examples of foreign bodies from the appendix, apart from concretions, not a few of which owe their preservation to the assiduity of Mr. W. H. Battle.

Pellet of Solder (No. 2283'1, Roy. Coll. Surg.).—An irregular pellet of solder 1'2 c.m. ($\frac{1}{2}$ in.) in extreme diameter, which was taken, after death, from the distal end of the vermiform appendix. The appendix

was throughout healthy. From a patient (a soldier) who died of uncomplicated typhoid fever. The solder was probably derived from the tinned beef which was served as rations.

Fragment of "Tin" (No. 2282'1, Roy. Coll. Surg.).—The terminal portion of an appendix, in the end of which there is loosely impacted an irregular fragment of tinned iron, apparently from a "bully beef" tin. The appendix was taken after death from a soldier who succumbed to extensive lacerated wounds of the shoulders, January, 1916. It presented no signs of inflammation.

Iron Nails (No. 2286'1, Roy. Coll. Surg.).—A vermiform appendix. Tightly impacted in its distal end, there are two small iron nails and a pin. The heads of the former lie downwards. The pin, which has been bent into a hook for the purpose of being swallowed, has advanced with the bend forwards. The appendix was successfully removed by Mr. W. H. Battle, from a girl, aged 17, who had suffered from repeated attacks of pain in the right iliac fossa.

It may be remarked, in passing, that when pins are intentionally swallowed, they are usually bent in the way mentioned (No. 2251'1, Roy. Coll. Surg.).

Grape-seeds (No. 1106E, St. Thomas's).—Three grape-seeds which were found in a vermiform appendix after excision. The appendix was removed from a woman, aged 23, whose attack of appendicitis, the first, commenced three days before admission, with abdominal pain, vomiting, and diarrhoea.

Bristles (No. 1106D, St. Thomas's).—A collection of pale, stiff hairs which were found in a vermiform appendix removed by operation. The patient had had several attacks of appendicitis; he wore no moustache. As he was a photographer's assistant, the hairs were possibly introduced by a habit of sucking brushes.

Shot (No. 1106B, St. Thomas's).—Two small shot which were taken out of an excised appendix. From a girl, aged 19, with a history of six attacks of appendicitis; the first, fifteen months previously. The appendix was long, and *slightly kinked* by the mesentery. The shot were embedded in soft faecal material. The patient was in the habit of eating game fairly frequently.

Fig-seeds (No. 2279'1, Roy. Coll. Surg.).—Eight *fig-seeds* which were found mixed with faecal material, in a vermiform appendix removed by operation. From a gentleman, aged 35, who had suffered from an attack of appendicitis two and a half years previously. He was on that occasion confined to bed for about a fortnight. Since the attack, the

patient often experienced pain and discomfort in the right iliac fossa, after exertion. He was in the habit of taking considerable quantities of figs.

In the course of the 125 examinations of appendicular contents (excluding concretions) to be referred to later, I came across the following foreign bodies in three—viz., a slender piece of fish bone; a short fragment of black bast fibre of the kind used for stiff brooms or brushes; one or two human hairs of medium length.

Into the same category, of course, would fall foreign bodies which have escaped into the abdomen after perforation. The earliest example of this in the College collection, and one, therefore, of historical interest, is that observed by Joseph Swan, F.R.S. (*b.* 1791, *d.* 1874).

(No. 2280'1, Roy. Coll. Surg.) The end of a small intestine with part of the cæcum and of the vermiform appendix. A rod of white glass has been passed from a perforation in the appendix, into the cavity of an irregular abscess in the surrounding tissues. The perforation is stated to have been due to the presence of a seed. The patient was a young man, who died from peritonitis following the rupture of the inflamed structure.

According to their construction, intestinal concretions admit of being divided into two groups; the differentiation between which is most pronounced in the case of the horse, and which may be here indicated by reason of its bearing on the present subject. Some of the intestinal concretions from this animal are hard, compact, finely laminated calculi, composed chemically of ammonio-magnesium phosphate, or of this mixed with a varying proportion of calcium phosphate. They are invariably furnished with a small foreign body of some kind which acts as a nucleus. My own microscopic examinations of the compact, concentrically laminated districts, carried out by means of sections made by the petrological method of cementing and grinding, show that the transparent substance of the lamellæ is crossed by crooked lines, having a radial direction, and indicating that the material is a close aggregate of long, interwoven and somewhat coarse crystals. This is true of areas where no radial marking is detectable with the naked eye. When fragments are decalcified with HCl there is disclosed, as has long been known, an animal matrix which retains the original form. The source of the magnesium phosphate is usually attributed to its presence in the wheat, oats, or hay, &c., ingested. In confirmation of this, it has been pointed out that enteroliths are never observed in carnivora; and that

they occur less frequently in omnivora than in herbivorous animals. The name "enterolith" should be restricted to such proper forms of calculi.¹

From the pathological standpoint the question may be raised, in passing, whether the objects known as coprolites might represent intestinal calculi. Apart from the general evidence that coprolites are fossilized intestinal excreta, arising from their great numbers and distribution, I submit that the structural differences between the two would, without any other proof, be decisive. Enteroliths invariably exhibit both to the naked eye, and in microscopic section, the concentric striation (alternating with, or even crossed by a radial) common to all calculi wherever formed. The only trace of internal structure recognizable in the coprolite, is the thin spiral line which is found in those of Saurian origin; the spiral configuration may likewise be indicated externally by the presence of a shallow groove; proving that the terminal part of the gut of such reptiles was furnished with a spiral valve of the kind present in certain living forms of fish. In the shark, e.g., such a valve extends for a considerable length of the large intestine; in the dog fish, the valve is limited to its terminal part. In a particularly fine coprolite in my possession the spiral line is so remarkably clear in the polished cross section, in consequence of its having been, in places, filled in with iron pyrites, that I venture to figure it. Microscopically, it exhibits no trace either of concentric or of radial striation.

Mr. R. H. Burne and I were able to reproduce the thing artificially by driving soft clay along the intestine removed from the recently dead dog-fish. The spiral valve in this fish ceases a few inches from the vent, the terminal portion of the gut being straight. Somewhat soft, white modelling clay was forced along the intestine with a syringe, as far as, and into the straight valveless portion, but only so as to partially fill it; a second mass of clay, coloured blue with French ultramarine, was now introduced from above, and driven onwards so as to completely distend the terminal segment of the gut. The parts were then allowed to become quite dry. The dried intestine having been peeled off, a transverse section of the lowest mass disclosed a thin outer layer of white clay, and a central mass of blue, in which there were segments of a fine spiral of white.

¹ It may be pointed out, as a matter of nomenclature, that the word "concretion" is of wider meaning than that of "calculus," and does not necessarily imply any inorganic deposition, as, for example, in the case of hair-balls. "Enteroliths," or proper calculi, comprise those formed in any part of the intestine, or in the stomach.



FIG. 18.

A polished transverse section of a coprolite, showing the spiral line (filled in places with iron pyrites), indicative of the action of a spiral valve with which the large intestine must, therefore, have been furnished. There are no traces of radial or of lamellar striation. (Natural size.)

Sectio coprolithi transversa spiram monstrans causatam valvulae intestini actione. (Magnitudinis naturalis.)



FIG. 19.

A cross section of a dried clay mass from the lowest, straight part of the gut of a dog-fish, obtained by forcing soft modelling clay along the intestine. The spiral disposition so shown closely reproduces that in the coprolite. (Twice the natural size.)

Scyllium canicula: Sectio transversa spiram monstrans in argillâ quae in intestinum rectum desuper pulsa erat. (Bis magnificata.)

Coprolites not infrequently (as in the case of that figured) include fragments of ingested bone (which have, in process of time, acquired a deep black colour); as already stated enteroliths are unknown in carnivora, and but rarely seen in omnivorous animals.

The second form of equine concretion contains a conspicuous proportion of organic *débris*, the residues of vegetable ingesta; largely, but not exclusively oat-hairs; like proper enteroliths, they may attain a notable, though not so extreme a size.

The term *stercolith* should be reserved for such mixed forms; amongst farriers they are known as dung balls. Their exterior, in place of being smooth, is regularly, and coarsely tuberculated and in section the phosphatic deposition does not form regular concentric lamellæ, but has an extremely coarse, somewhat undulatory disposition in ill-defined zones which are incomplete or interrupted in their circuit; the various spaces in the section, like the pits between the external tubercles, are filled with vegetable *débris*. It must be admitted, nevertheless, that the proper enteroliths are not infrequently admixed with intestinal *débris*, although still constructed after a different manner from those last described. Thus, besides the typical forms where the entire structure is compact and produced directly upon a foreign body, there are others in which a large well-defined area immediately around the nucleus is constructed of coarse, radially set, crystalline columns, with wide intervening fissures filled with *fæcal débris*; whilst in the calculus beyond there are not unusually two classes of broad, alternating, sharply defined zones; one, compact and concentrically striated, and the other coarsely striated in the radial direction, and of somewhat open texture; furnished with narrow radial clefts which are occupied with intestinal *débris*. And what is more, in microscopic preparations made from the most unequivocal and compact equine enteroliths, after decalcification with HCl, a certain number of fragments of plant tissue are invariably disclosed. Like the normal contents of the colon the plant *débris* included in enteroliths gives no cellulose reaction with iodine solution followed by 75 per cent. H_2SO_4 , the cellulose element having undergone bacterial digestion.

In one of the proper equine enteroliths in the College collection (S.2) the section displays a single broad zone in the midst of the body, which has the typical construction (or want of construction) shown by the *stercolith*—i.e., a coarse, somewhat ill-defined, undulating belt, devoid of concentric or radial striation, and honeycombed with cavities containing vegetable *débris*, almost entirely of oat-hairs.

There is a second, almost identical; and in another (S.64), still more curiously, one hemisphere is constructed after the manner of a compact enterolith; and the other, as a typical stercolith, with a coarsely tuberculated exterior and no regular internal structure. And as a final combination, there is a stercolith formed upon a proper, compact, doubly striated enterolith the size of a hen's egg (P.6,c).

Oat-hair Concretions.

The objects met with in the *human intestine* which most resemble the stercoliths of the horse, are the concretions resulting from the accumulation of oat-hairs. One of the first recorded examples of such is contained in the College Museum, and is sufficiently interesting to cite briefly for its bearing upon appendicular concretions. The specimen (No. 2346'1, Roy. Coll. Surg.) consists of three concretions of irregular form, removed after death from the transverse colon. Of one, a section has been made: this concretion, which in its chief diameter measures 5 cm. (2 in.), is coarsely laminated, and consists of alternating zones of a fine, pale yellow felt, and phosphatic material; in its centre there is a plum stone which serves as a nucleus. The felt consists, as told microscopically, of the small stiff hairs attached to the summit of the oat. The free surface is, for the most part, invested with a thin layer of phosphate. An account of this case is published in the *Transactions of the Royal Society*, 1822, by Mr. Children. The patient, a Lancashire carpenter, aged 19, was in the habit, during the hot weather of July, 1814, of eating unripe plums and swallowing the stones. Some months afterwards he began to suffer from abdominal pain and diarrhœa. A hard, circumscribed mass was discovered on one side of the abdomen. After death, the three concretions were found closely compacted together in the transverse colon. His usual diet was milk porridge twice a day—viz., at breakfast and supper, the milk being thickened with oatmeal. His dinner usually consisted of meat and potatoes, with oatcake. In the afternoon he ate oat-bread and cheese; so that he never took a repast without oatmeal in some form. Chemical analysis showed the inorganic material to consist of calcium phosphate and ammonio-magnesium phosphate.

The recognition of vegetable hairs in such concretions was due to Dr. Wollaston, Mr. Clift, the first conservator of the College Museum (1800-49) having suggested that they were from the oat,

which proved to be the case.¹ In the first accounts the hairs are described as pointed at both ends: this, although true to fact, is due to their slightly expanded, obliquely set bases having been broken off. In section, as told by the naked eye, they consist of a close felt, of pale yellow or fawn colour, which microscopically is composed of stiff, sharply pointed, vegetable hairs. These, as already stated, are the setæ which project from the summit of the oat. If the ripe, dried oat is dissected under water, after maceration in dilute alcohol, on removing the pair of unequal glumes, the summit will be seen to bear two short processes which are the stumps of the withered

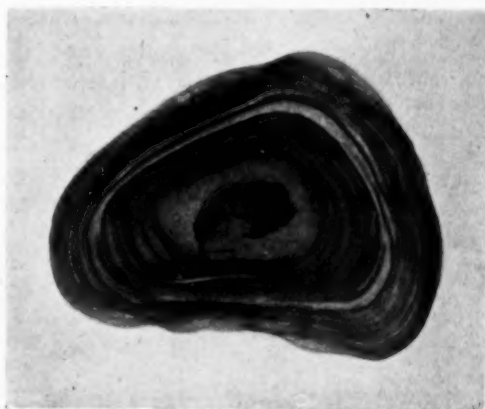


FIG. 20.

A skiagram of half of the largest of the oat-hair concretions from the case ("Chambers") referred to in the text, showing the alternating translucent zones of oat-hairs, and the opaque zones of calcium and ammonio-magnesium phosphate. In its centre there is a plum stone which serves as a nucleus. The laminar alternation in this concretion is particularly well pronounced. (Roy. Coll. Surg.) (Natural size.)

Concretio humana, bisecta, avenae fructus pilis constructa, e colo post mortem extracta. Monstrat pictura radiographica zonas pilorum translucidas aliis intermixtas opacis, ex calcii et ammonio-magnesium phosphate constantibus. In concretionem mediâ monstratur fructus nucleus (Prunus domestica). (Magnitudinis naturalis.)

styles, and from the summit around, as well as from the sides, there project a conspicuous number of stiff hairs set with a parallel direction

¹ Marcet, "Essay on Calculous Diseases," 1819, p. 139.



FIG. 21.

The fruit ("seed") of the oat on the left-hand side, and of the wheat on the right, enlarged about three times, showing the stiff, parallel hairs which arise from the summit and sides of the oat, and from the summit only of the wheat. The two stumps on the apex of each are the remains of the styles.

Avenae (in parte sinistrâ) et tritici (in parte dextrâ) fructus, setas monstrantes quibus praediti sunt. Utriusque apud summam stylorum duorum residua projiciuntur. (Ter magnificantur.)

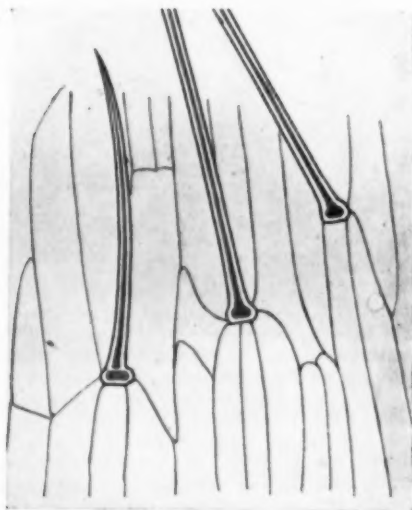


FIG. 22.

A piece of the epidermis stripped from the pericarp of the oat, about the summit, showing three thick walled hairs, the expanded bases of which are intercalated between the elongated cells composing the epidermis; two are shown only in part. (3 obj.)

Epidermidis lamella de avenae fructu detracta, trium setarum monstrans bases inter cellulas insertas.

upwards. If the epidermis which invests the pericarp is detached, the hairs come away with it, their expanded bases being intercalated between its elongated cells. The hairs are thick-walled, sharply pointed at the free end, and have an expanded, oblique base by which they are securely fixed between the general epidermal cells. The obliquity of the base allows of the hairs being directed vertically in spite of their fixation at the sides of the fruit. A microscopic examination of the porridge made either from the coarse or the finer kind of oatmeal will always disclose a certain number of the hairs, or their fragments; which, being composed of cutine, resist peptic and tryptic digestion; as well as the bacterial digestion which takes place in the large intestine of the human subject.

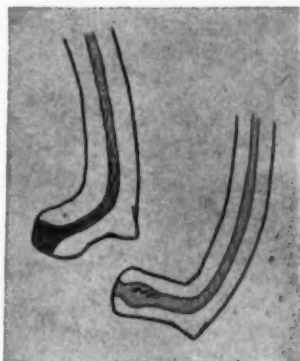


FIG. 23.

The bases of two of the stiff, thick-walled hairs from the pericarp of the oat. The obliquity of the base is pronounced; the internal cavity opens through its centre; the wall of the base is devoid of the canals which are present in the hairs from wheat. ($\frac{1}{8}$ obj.)

Avenae fructus setarum duarum bases, obliquae atque paululum expansae.

Very similar hairs occur on the wheat grain, although they are not so long, and consequently do not arrest attention; they are limited to a broad area at the summit, which presents, centrally, the stumps of the two withered styles. Microscopically the hairs differ from those on the oat, in that the expanded and oblique base is traversed by a few short canals. A microscopic examination of wheaten flour, even of the finest steel-milled kind, will show the presence of a certain number of the hairs, either intact or fragmentary. By the difference referred to, it is easy to recognize the presence of both kinds of hairs in appendicular concretions.

The oat-hair concretions in the College collection, of which the location is recorded were, as might have been anticipated, from the large intestine, since only the indigestible elements of the food are allowed by the ileo-colic sphincter to pass through the ileo-colic valve; and in the small intestine such hairs would be distributed through more diffuent contents and less liable to aggregate. Of the eighteen examples from different cases, in the human subject, in seven it is recorded that the concretions were either removed from, or passed by, the rectum. In specimen I.10, however, a collection of such concretions was found in the ileum; and the following is an additional example of a similar location:—

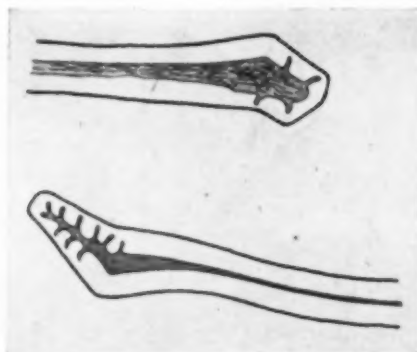


FIG. 24.

The bases of two hairs from the summit of the wheat fruit, showing the oblique, expanded base, the wall of which is traversed by a few short canals, and in this differs from the hairs of the oat. ($\frac{1}{4}$ obj.)

Tritici fructus setarum duarum bases; obliquae, paululum expansae, atque canalibus paucis perforatae.

(No. 2348.1, Roy. Coll. Surg.) The concretion, which has a chief diameter of 4.5 cm. ($1\frac{1}{4}$ in.) was successfully removed from the small intestine of a boy who had for three years suffered from abdominal pain, lately accompanied with recurrent hæmorrhage from the bowel: at the operation the concretion was found in the small intestine, and lying close to the liver: it was removed through a longitudinal incision the bowel being afterwards sutured. The boy, who was employed in a poulterer's, was in the habit of wetting his fingers in his mouth when plucking birds, and in the College catalogue it was stated that

the concretion consisted of feather down: a re-examination shows that this is not the case, but that the material is solely of oat-hairs.

From a study of the entire series from the human subject, eighteen in number, the following general statements may be deduced. As to number, they may be single; or multiple, three or more; in one case the patient had voided several smaller, before the one which is preserved, was extracted *per anum*. In form they may be spherical, oval, lenticular, irregularly tuberculated, pyramidal: all are represented. The largest, which is pyramidal in form, measures 6 cm. ($2\frac{3}{8}$ in.) in chief diameter. The pyramidal are distinctly faceted, and were evidently components of groups. In one instance two appear to have become cemented together. They are, if not admixed with phosphate, extremely light, and of pale brown or fawn colour. In nearly every instance a fruit-stone has served as a nucleus upon which the deposition has taken place. In section they may present no trace of laminar or other structure; or they may be laminated from the interposition of concentric lines of phosphatic material. The magnesium phosphate, which is generally present in the inorganic constituent is probably derived from the vegetable ingesta.

Concretions of oat or of other vegetable hairs have been taken from the alimentary canal of various kinds of animals; as illustrated by numerous specimens in the College collection.

As will be pointed out later, the common form of appendicular concretion consists solely of vegetable residua; but I have not come across any, and know of none, constructed solely of oat hairs, and so, comparable with those met with in the colon; although such hairs occur at times in conspicuous numbers in concretions from the human appendix, as they do in the equine stercoliths already described. The felting of the hairs which is so striking in oat-hair concretions can only be matched by that of the animal hairs which compose the hair-balls from the stomachs of ruminant cattle, like the goat and ox, and which arise from the habit these animals have of licking their coat. In the concretions of human hair from the human stomach, felting is rendered impossible by the length of the hair. The process is shown to a lesser degree than in ruminants, in a huge hair-ball from the College collection (O.62), which was taken from the stomach of an alligator found in a dying condition on the bank of the Amazon, Brazil. It measures 30.5 cm. (12 in.) in diameter, and consists of loosely felted stiff animal hairs. The hairs were not identified, though, from a comparison with those of the capybara, it was clear they did not come

from that animal. But the two most perfect examples of felting in the College museum are the following:—

(No. 2298'1, Roy. Coll. Surg.) Two light, compact masses of felt, passed, with others, by a boa constrictor which swallowed the blanket usually supplied as a covering. They are accurately articulated, and the facets on the free end of each show that further formations were passed with them. The upper is 6.5 cm. ($2\frac{1}{2}$ in.) in its longitudinal axis; the lower about half of this. In the horizontal plane each is circular, about 7.5 cm. (3 in.) in diameter, and is moulded to the form of the intestinal canal.

(No. 2258'1, Roy. Coll. Surg.) A somewhat oval, buff-coloured concretion, 13 cm. (5 in.) in chief diameter. It is extremely light, and consists solely of paper pulp. The concretion was taken from the stomach of a goat that had been killed for consumption, the animal being fat and healthy. The donor stated that the goats at Kalgoorlie (Australia), where the animal was killed, are particularly fond of eating paper.

Enteroliths: Human.

In the human subject proper intestinal calculi, or enteroliths, formed within the lumen of the *intestine*, are very rare. There are but three in the College collection, and two of these have been produced upon nuclei. They are as follow:—

(No. 2349'1, Roy. Coll. Surg.) Half of an oval calculus, 3.8 cm. ($1\frac{1}{2}$ in.) in chief diameter, which was removed from the rectum. In its centre is a plum-stone upon which the concretion has formed. As shown in the divided surface it is constructed of a series of closely apposed, radially set, crystalline cones, which are regularly crossed by fine transverse striæ, the whole appearance closely resembling that of a calculus of calcium oxalate, the deep brown colour of which it also reproduces. Its exterior is lowly nodulated, or granular, from the projection of the growing bases of the cones; scattered over it are a certain number of crystals which consist of ammonio-magnesium phosphate, their unusual whiteness (or efflorescence) being due to loss of the water of crystallization. Chemical examination shows that the calculus consists solely of magnesium phosphate, without any trace of calcium oxalate. The calculus was extracted from the rectum of a man, aged 50, who complained of constant diarrhœa and frequent desire to evacuate the bowel; it was withdrawn, though with some difficulty, by means of stout lithotomy forceps. The specimen is briefly recorded,

but without chemical examination, by Mr. E. H. Fenwick.¹ This, in the human subject, is so extremely rare a form of enterolith that I venture to insert a figure of it. It stands alone in the unique collection

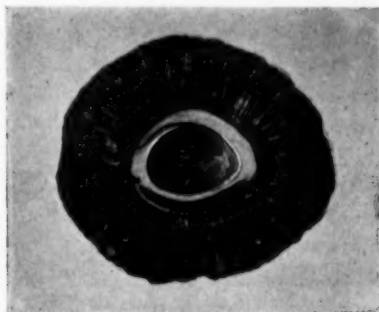


FIG. 25.

The human intestinal enterolith of magnesium phosphate, referred to in the text, showing its laminar and radially crystalline construction, and its granular or tuberculated surface. In its centre there is a plum-stone which has served as its nucleus. (2349-1, Roy. Coll. Surg.) (Natural size.)

Enterolithus per hominis anum extractus. E magnesi phosphatæ solum constat ut calculi quidam in animalium intestino nonnunquam reperti. In concrezione mediâ includitur fructus nucleus (*Prunus domestica*).

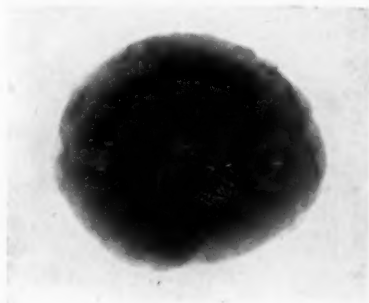


FIG. 26.

A skiagram of the same enterolith; like calcium phosphate, magnesium phosphate filters off the rays. (Natural size.)

Ejusdem enterolithi pictura radiographica. Per magnesi phosphatæ ex quâ calculus solum constat radii vix percolantur. (Magnitudinis naturalis.)

¹ *Trans. Path. Soc.*, 1886, xxxviii, p. 261.

of calculi in the College Museum, which contains no less than sixteen examples of this kind from the lower animals. These have the same coloration and intimate structure, though externally they are more coarsely tuberculated. The regularity, coarseness, and flattened character of the tuberculation have led to these concretions being named "Rhinoceros bezoars." The term "bezoar," which is of Eastern origin, has been applied to every kind of gastric or intestinal calculus coming from the lower animals. Those of magnesium (di)phosphate in the College, are spherical or oval in form, and range from the size of a golf-ball to that of a tennis-ball. Some of them (T8, 9, 10, 14, 16) show a nucleus—viz., an acorn, the remains of some seed, a pebble, a pebble, a small mass of vegetable matter. They are probably all from herbivora, or omnivora, the magnesium phosphate being furnished by vegetable ingesta. The object of excluding calcium oxalate from its composition is that enteroliths of this substance likewise occur in herbivora, and as I have long since pointed out, the discovery of such in the human subject may be anticipated in consequence of the spread of vegetarianism; for the calcium oxalate in such enteroliths is primarily derived from that contained in vegetable ingesta. The enteroliths of calcium oxalate in the College, resemble structurally, and in their external characters, the smooth, polished forms of similar calculi from the pelvis of the kidney or urinary bladder, or from cysts within the renal substance; and display on section, to the naked eye, a concentric lamination, the lamellæ, as told in microscopic sections, being composed of cones of long transparent crystalline forms, the bases of which correspond with the external limit of the lamellæ. In all of those in the College (five in number), there is a conspicuous nucleus consisting of vegetable debris—fibre and hairs.

The finest example comes from the collection of Sir Hans Sloane and was catalogued as "An Elephant Bezoar": it is almost spherical, 12 cm. ($4\frac{3}{4}$ in.) in diameter, and of a pale fawn colour. The largest of all is chemically less pure, and contains only 66 per cent. of calcium oxalate, the rest being chiefly calcium carbonate: of oval form, it measures in the longer axis 16 cm. ($6\frac{1}{2}$ in.)

The second human enterolith in the College is: (No. 2606A, Roy. Coll. Surg.). An irregularly oval calculus 5 cm. (2 in.) in chief diameter, which was removed from the rectum of a lady, aged 69, from whom a concretion had previously been extracted. The large central cavity now present in it, was filled with a crumbling material mixed with large glistening crystals (ammonio-magnesium phosphate), some

of which still project from its sides. The peripheral, remaining, part which has a thickness of 7 mm. is of a light brown colour, somewhat coarsely laminar, and crossed by the radial striæ indicative of crystalline

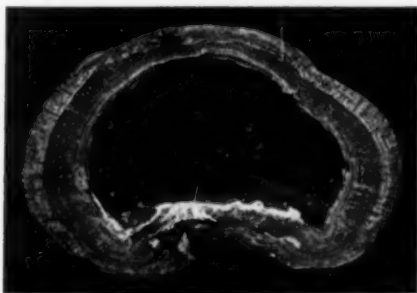


FIG. 27.

The second human enterolith (2606A, Roy. Coll. Surg.) described in the text. (Natural size.)

Enterolithus per mulieris anum extractus. Ex ammonio-magnesii phosphate cum calcei phosphate commixtâ constat. Calculus substantiâ fragili et crystallis magnis intus impletus est. (Magnitudinis naturalis.)



FIG. 28.

The third enterolith described in the text; consisting of large crystals of ammonio-magnesium phosphate, formed upon part of a tooth which had been accidentally swallowed many years previously. (2350-1, Roy. Coll. Surg.) (Natural size.)

Enterolithus sive calculus intestinalis per anum dejectus. Ex crystallis ingentibus ammonio-magnesii phosphatis construitur. In calculo medio monstratur unus e dentibus incisoribus quem glutiverat aeger. (Magnitudinis naturalis.)

structure. It is composed partly of organic matter, the inorganic consisting of ammonio-magnesium phosphate, with a trace of calcium phosphate: no oxalate is present.

The third and last example in the College Museum (No. 2350.1) is: An intestinal calculus 4.5 cm. ($1\frac{3}{4}$ in.) in its chief diameter, which was passed *per anum*. Its exterior is highly irregular and composed of large crystals arranged in coarse fasciculi devoid of any orderly disposition. Portions have been removed to display the fragment of an incisor tooth which forms the nucleus of the concretion. The crystals consist of ammonio-magnesium phosphate, their whiteness being due to the loss of water of crystallization. The calculus was passed naturally by a woman, aged 70, who had for many years suffered from constipation. The presence of the tooth was explained by the fact that sixteen years previously one of her upper central incisors had been broken off, and that she had swallowed the fragment; the stump of this tooth was still in position.¹ The chemical composition of this calculus, ammonio-magnesium phosphate, is, like that of the two preceding, matched by some of the equine and other animal forms; the source of the magnesium phosphate or of the ammonio-magnesium phosphate being furnished by vegetable ingesta: the inorganic salts of the succus entericus amount only to 0.8 per cent. of the secretion, and would be inadequate to produce such voluminous calculi.

Appendicular Concretions.

Passing on now to appendicular concretions. These may be divided into (1) enteroliths, or proper calculi; and (2) stercoliths, or phosphatic depositions admixed with faecal material. Biliary calculi which have entered the appendix have, of course, to be eliminated from the true forms. In the College Museum there is an excised appendix (presented by Dr. H. A. Lediard) which contains two such calculi, about the size of hemp-seeds, and of deep black colour; a third was used to confirm, chemically, the macroscopic diagnosis. The appendix was removed in an acutely inflamed and gangrenous condition, from a man aged 47. The donor had previously removed another appendix, which was found to contain some turbid mucus and eleven small calculi of varying sizes, from that of a pea to the head of a large pin; they were black in colour, somewhat soft, and faceted, and chemically consisted of cholesterin and bile pigment.² Such an occurrence is alluded to by several writers.

¹ H. W. G. Mackenzie, *Trans. Path. Soc.*, 1892, xliii, p. 70.

² H. A. Lediard, *Lancet*, January 12, 1907.

In his "Treatise on Cholelithiasis" (1892; and New Sydenham Society's translation, 1896), Naunyn has given all the cases recorded up to that date. Dr. Lediard's two are additions to the list, as is also that recorded by Mr. Mayo Robson, in which perforation of the appendix followed the lodgement of a gall-stone in it.¹ Such calculi might, of course, serve as nuclei for proper appendicular concretions.

Appendicular Enteroliths or Proper Calculi.

Of a true enterolith or proper calculus from the appendix—unmixed, i.e., with faecal material—I know of no pure example; nothing, that is to say, strictly comparable to the enteroliths met with in the human intestine, or in that of the lower animals. The sole specimen which I have had the opportunity of examining is the following, and, as will appear, it is of a mixed kind: centrally, stercolith; peripherally, enterolith. A reference to the equine forms of intestinal concretions already described brings out the interesting fact that amongst these, mixed forms may be met with; in the College collection there are two such in which a broad stercoraceous and partially calcified zone occurs in the mid substance of a proper compact calculus or enterolith; and a third, in which one hemisphere has the structure of an enterolith, and the other that of a stercolith.

The specimen from the human appendix (No. 2559E, Roy. Coll. Surg.) is half of a somewhat oval calculus, 1.2 cm. ($\frac{1}{2}$ in.) in longer diameter, which was taken out, together with a second of about the same size, from an excised appendix, the wall of which was thickened and indurated from chronic inflammation. Its external surface is irregularly nodulated, and the consistence unusually hard. In the median section there is displayed a double structure. Centrally it is of loose crumbling consistence, and has the imperfect lamination and dirty white colour of the common appendicular stercolith. Beyond this oval centre the structure is hard, very compact, and of pure white, with a reflecting or polished surface; and it exhibits a regular, close concentric or undulating striation corresponding with the elevations or tubercles which project from the exterior. The demarcation between the two parts is fairly abrupt; the second deposition is thinner at the poles than elsewhere. No nucleus is present. Scrapings of the friable central portion yield CO_2 when treated with HCl beneath the microscope, the residues consisting of plant debris.

¹ *Lancet*, December 29, 1906.

A chemical examination of the second calculus, by Mr. L. S. Dudgeon, showed it to contain as bases, calcium and magnesium; and as acids, carbonic, phosphoric, and oxalic. An inquiry into the usual dietary of the patient, a policeman, showed that he was a hearty consumer of meat and vegetables, but not a vegetarian.

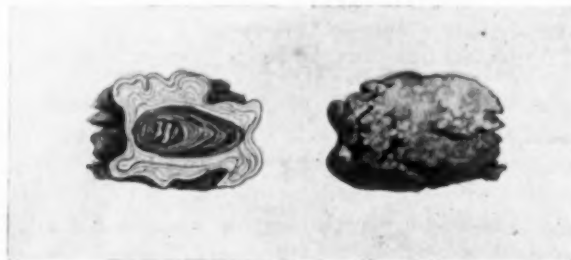


FIG. 29.

(A) Half of the mixed specimen of stercolith and enterolith described in the text. Centrally it is of crumbling consistence, imperfectly laminated, and has the characters of a common stercolith. Beyond this the structure is hard and compact, and is marked with an undulating lamination in correspondence with the tubercles on the exterior. Chemically it consists of calcium and magnesium bases, in combination with carbonic, phosphoric, and oxalic acids. (B) As viewed exteriorly. (Enlarged twice.)

Appendicis calculus sive enterolithus generis raro reperti. In concretionem mediâ structura ex calcii salibus constat cum substantiâ faecali, ut solet, commixtis: structura autem aliter, bene compacta, alba, atque laminata. Calculi superficies, tuberculata. Scrutatio chemica demonstrat bases, calcium et magnesium; acida, carbonicum, phosphoricum, oxalicum. (Bis magnificatur.)



FIG. 30.

A skiagram of one half of the same appendicular concretion. The salts in the compact periphery have filtered off the rays. (Natural size.)

Ejusdem enterolithi appendicularis pictura radiographica. (Magnitudinis naturalis.)

Appendicular Concretions: Multiplicity and Articulation.

The multiplicity and articular facetting, so familiar in biliary calculi, are but seldom met with in appendicular concretions. I can recount some four examples, two of the concretions from the first of which are figured below.

A second instance was furnished by an appendix removed after death: the two concretions it contained were articulated end to end, and measured 0.6 cm. transversely, and 2.5 cm. in their combined length. They are again referred to in the group of concretions without nuclei.¹

A third example is No. 1107N., St. Thomas's Hospital, an appendix excised from a woman aged 30, who had suffered from several attacks of abdominal pain, which invariably started in the epigastrium and was attributed to indigestion. When admitted there was an acutely tender spot in the right iliac fossa, the suprajacent skin being hyperæsthetic. Its distal half is dilated and partly filled with two laminated concretions which are in contact; others have fallen out from the dilated portion beyond.

And the fourth instance is No. 1107C, St. Thomas's: a vermiform appendix in which lie three articulated concretions, the largest 1.5 cm.



FIG. 31.

Two of a group of four faceted appendicular concretions; the laminated section shown highest is that of the lesser of the two below. Their biliary nature was excluded by chemical examination. They were from the partly gangrenous appendix excised by Dr. H. A. Lediard, from a girl, aged 8, and are again referred to in the group of concretions devoid of nuclei. (Natural size.)

Appendicis concretiones articulatæ. (Magnitudinis naturalis.)

($\frac{3}{8}$ in.) in chief diameter. During its excision it was found extensively adherent to the small intestine.

Multiplicity does not, of course, necessarily involve contact, and so, articular facetting.

In some cases, lastly, a mesial section will show that the concretion is compound—i.e., has been formed upon more than a single centre.

¹ The most remarkable instance of multiplicity accompanied with facetting, amongst urinary calculi is, perhaps, in St. Thomas's Hospital Museum. The calculi are 137 in number, and others had been passed spontaneously. They were removed suprapubically from a man aged 75, and consist of ammonium urate. The largest is 1.5 cm. in diameter.

Appendicular Concretions: Fracture.

The occurrence of fracture in an appendicular concretion, during life, I have met with but once; and that, in a concretion of the common kind, but remarkable for its length. The appendix was excised from a male patient whose symptoms, violent general abdominal pain, dated from the preceding day. There were flakes of lymph on the peritoneum; the concretion lay in the distal end, and had not led to perforation. The fissures, shown in the accompanying figure, were disclosed only after the surface had been gently scraped away under water, in the usual way I adopted for all such examinations; and they reached



FIG. 32.

The concretion described in the text, showing an incomplete fracture which has occurred during life. The fissures, which penetrated almost to the centre, are, for the sake of clearness, shown as dark lines, but were really filled with a soft white material which microscopic examination showed to be largely bacterial. The concretion had been placed in formol solution immediately after its removal. The fracture was disclosed only after the removal of the surface, which was carried out as usual by gently scraping the concretion under water.

Appendicis concretio insigniter elongata, atque in parte fracta. Fissurae omnes substantiâ impletæ sunt molli et albâ e bacteriis præcipue constanti. (Magnitudinis naturalis.)

almost to the centre. They were accurately filled with a soft white material which microscopic examination showed to consist of bacteria associated with a finely granular basis. The concretion was devoid of any differentiated nucleus. The fracture in this case cannot rightly

be called spontaneous, for it was doubtless due to some external pressure, or to some sudden flexion of the appendix; and its occurrence from such a cause must, apparently, be connected with the unusual length of the concretion.

The spontaneous fracture of urinary calculi is a phenomenon too well known to need more than mention,¹ and is attributable not to compression, but to the physical changes, swelling or shrinkage, of the matrix or animal basis of the calculi, which leads either to delamination, or to their disruption in wedge-shaped fragments, for both forms of fracture occur. An occurrence more akin, however, to that which has taken place in the appendicular concretion, is the fracture of a calculus in the gall-bladder, of which a very few instances have been recorded. There is one such in the museum of St. Thomas's Hospital (No. 1401): A gall-stone with six fragments of the same, taken after death from the gall-bladder; the edges of the several pieces are smoothly rounded, showing that the fracture took place some time before death. From a woman, aged 66, who had complained occasionally of pain and tenderness in the right hypochondrium. Here the comparatively exposed position of the gall-bladder, and the somewhat friable character of the calculus concur to render such a result possible from external violence.

Appendicular Concretions: With Nuclei.

In conducting examinations with the object of discovering a nucleus (when this is not obvious externally), the method adopted was to incubate the concretion, whether previously dried or not, in dilute alcohol, at 37° C., for many days, in a closed test-tube; and then to investigate it under water, by rotating it with one pair of forceps, and gently scraping or delaminating it with another, the whole proceeding being viewed through a lens. By this means, also, the consistence and degree of lamination, &c., can be observed; and microscopic preparations can be made at any stage of the procedure. The simple method of cutting through a concretion, especially if dried, in search of a nucleus, is unreliable for proving a negative, although it is, of course, effective if the nucleus is of a gross kind. The following series may serve to illustrate such as contain some foreign body as a nucleus. In the

¹ W. M. Ord, "Influence of Colloids upon Crystalline Form," p. 93, where references are made to cases also observed by others; S. G. Shattock, "The Microscopic Structure of Urate Calculi," *Proc. Roy. Soc. Med.*, 1913, vi (Path. Sect.), p. 1, where the occurrence of microscopic fractures, repaired by subsequent deposition, is referred to.

College collection there are two concretions, from different cases, which have formed upon pins.

Pin (No. 2355'1, Roy. Coll. Surg.).—A fusiform calculus 1.5 cm. ($\frac{3}{4}$ in.) which has formed upon a pin, and was removed after death from the vermiform appendix. From a child, aged 6, who was seized whilst at school, with pain in the abdomen, vomiting and diarrhoea; she was somewhat better on the day following, but died suddenly on the third day of illness. The appendix after death was found in a sloughing condition, and with the calculus shown within it. Douglas's pouch was filled with fluid fæces, which were shut in by adhesions of the omentum and small intestine.

Pin (No. 2356'1, Roy. Coll. Surg.).—An oval calculus, 2 cm. ($\frac{4}{5}$ in.) in its longer diameter, from a vermiform appendix; in its centre there is a pin upon which the concretion has formed. The appendix was removed from a man, aged 25, who was admitted with a twenty-four hours' history of acute pain in the right iliac fossa, accompanied with vomiting. It was found, at the operation, to be gangrenous and perforated; its apex was firmly buried in the lumbar muscles, and in this was contained the concretion shown.

Pellet of Lead or Solder (No. 2357'1, Roy. Coll. Surg.).—A small oval, finely laminated concretion about the size of a pea, which was taken from an excised appendix. The upper end has been removed to expose part of a pellet of lead or solder which forms its nucleus, and makes up the chief bulk of the mass. Chemically the concretion consists of phosphate and carbonate of calcium. The appendix was removed from a man, aged 48, an electrician, whose only attack of appendicitis was that for which the operation was carried out. Although the encrusting material, which is in but small amount, is more finely laminated and more translucent than is usual in appendicular concretions, a microscopic examination shows that it does not consist solely of inorganic material (calcium carbonate and calcium phosphate), but includes a certain amount of plant débris.

Small Shot (No. 2559'1, Roy. Coll. Surg.).—Five small shot which were taken out of an excised vermiform appendix; they were embedded in a large fæcal concretion. The appendix presented an extensive gangrenous perforation.

Grain of Wheat (No. 1104, St. Thomas's).—A perforated appendix removed by operation from a boy, aged 8. The perforation was caused by the entrance of a grain of wheat, which, encrusted with fæcal material, formed a concretion rather larger than a pea. Death occurred from general peritonitis.

Seeds of Raspberry, Strawberry and Fig (No. 2354'1, Roy. Coll. Surg.).—This appendix has already been referred to as one containing multiple concretions (No. 1107N, St. Thomas's). Examination of the largest, 1.3 cm. in chief diameter, disclosed the presence of a nucleus proportionate in length to the oval form of the concretion, and consisting of a group of eleven closely packed fruit seeds, embedded in a scanty amount of dark brown faecal material; beyond which the concretion was of lighter colour, and laminar construction. Of the seeds, five were flattened, oval, and larger than the rest, the pattern of fine ridges on their surface proving them to be either from the raspberry or blackberry; of the others, five were flattened and reniform, and corresponded with those of the strawberry; the eleventh was pale yellow, oval, bluntly pointed, and from the fig. The appendix was excised from a woman, aged 30, who had suffered from several attacks of abdominal pain which invariably started in the epigastrium. When admitted there was an acutely tender spot in the right iliac fossa, the suprajacent skin being hyperæsthetic.

Fig-seed.—Dr. H. A. Lediard has recorded a typical example of a laminated stercolith formed upon a seed which was identified as that of the fig.¹

Grape-seed.—The same author sent to the College of Surgeons in August, 1915, a grape-seed which formed the centre of an appendicular concretion.

In the following three cases the nucleus consists of one or more human hairs, unintentionally or purposely swallowed. The hair or hairs in these specimens lie centrally, and correspond with the long axis of the concretion.²

Human Hair (No. 2353'1, Roy. Coll. Surg.).—Four minute white concretions, in intimate connexion with each of two of which there is a fine straight hair, the longer of which is 1.1 cm. ($\frac{3}{8}$ in.) in length. From an unmarried woman, aged 20. There was a history of recurrent

¹ *Lancet*, January 12, 1907.

² The hair casts or "balls" which form in the human stomach and result from the intentional ingestion of human hair, are well represented in the College collection, which contains four examples. One of these comprises human hair admixed with woollen material, the patient (a girl, aged 24) having consumed the vest which she wore, in addition to her own hair. There is a further specimen in St. Thomas's (944B), in which the hair extended a considerable way through the pylorus into the duodenum. Besides these there are in the College collection, a cast consisting of cocoa-nut fibre; one of bast fibre, string, shoe-laces, and hair; and one of woody fibre from an insane patient who was in the habit of gnawing her furniture.

attacks of pain in the region of the appendix, accompanied with pain and sickness.

Human Hair (No. 2353·2, Roy. Coll. Surg.).—A small, oval, dark concretion, with which two or three hairs (one of them 6·5 cm. in length) are intimately connected; to its surface, moreover, there adheres a small elongated seed. From a girl, aged 3½, suffering from general suppurative peritonitis, due to perforation; the concretion had not escaped from the appendix.

Human Hair.—In this case the concretion was examined after maceration in dilute alcohol, by careful delamination in water; the process was carried out until a centre about the size of a barleycorn was isolated, from one pole of which the end of a fine hair projected; the whole object broke in two, whereupon each piece was made into a microscopic preparation by washing it in glycerine and pressing it beneath a cover glass. Each preparation contained a piece of (the same) human hair.

In the systematic examination of appendicular concretions I have at times found fragments of hair embedded in the body of the concretion, without there being any such in the centre; but in the preceding instances the hair, from its position, may be regarded as having furnished a nucleus.

Fibrovascular Bundle.—An elongated concretion, 1·3 cm. in longer diameter, from a boy aged 2 years. It was easily disintegrated by gentle scraping under water, and was of the colour and consistence of pipe-clay. Throughout its substance there were sparsely distributed small red, and darker coloured particles, which microscopic examination proved to be vegetable parenchyma with pigmented cell contents. On reaching the centre, a colourless, filamentous structure was disclosed exactly in its long axis. This, as shown by microscopic examination, was a long piece of a fibrovascular bundle.

(To be concluded.)

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(COMBINED MEETING.)

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Sections of Psychiatry and Neurology.

Section of Psychiatry.

President—Major F. W. Mott, M.D., F.R.S.

(January 25, 1916.)

SPECIAL DISCUSSION ON SHELL SHOCK WITHOUT VISIBLE SIGNS OF INJURY.

Opened by the PRESIDENT.

"SHELL SHOCK" is a term applied to a group of varying signs and symptoms indicative of loss of functions or disorder of functions of the central nervous system, arising from exposure to forces generated by high explosives. The forces producing shell shock are most commonly generated by the explosion of large shells, but also by mines, aerial torpedoes, whizz-bangs, trench mortars, bombs and hand grenades charged with high explosives. In a large number of cases, although exhibiting no visible injury, shell shock is accompanied by "burial." Sandbags may be dislodged from the parapet of the trench and strike the individual on the head or spine, and cause concussion without visible injury, or the roof of a dug-out may fall in. While lying partially buried he may be subject to the inhalation of noxious gases, so that a combination of causes may account for the severer effects of shell shock.

Although there may be no visible sign of injury, yet from the point of view of compensation or pension the War Office authorities very properly regard shell shock as a definite injury. This fact is of considerable importance, for as in the case of pension or compensation

in traumatic neurasthenia under the Employer's Liability Act, the notion of never recovering may tend to become a *fixed idea*. The detection of conscious fraud is not easy in many cases of shell shock in which recovery may be reasonably expected, for it is difficult in many cases to differentiate malingering from a functional neurosis due to a fixed idea. The first point is to be sure of your diagnosis that the disease is altogether functional, and being satisfied thereof to avoid all forms of suggestion of non-recovery. The second point is to strive against auto-suggestion of non-recovery by taking the man's mind off himself with amusements, games, and occupation, if possible in the open air. Prolonged massage and electricity of all forms are better avoided, as a rule, unless as a means of suggestion of cure by their use. Look cheerful and be cheerful should ever be the mode of greeting these patients.

At the Neurological Section of the Fourth London General Hospital exceptional opportunities have occurred for studying the functional neuroses, and to a less degree psychoses arising from shell shock. A considerable number of cases have occurred in which shell shock has been associated with visible injury. In a large number of cases there may have been physical concussion from the patient having been struck by sandbags when the parapet was blown down. Again, the explosive force may blow the man up into the air, and he may fall heavily on his head or spine on the ground, and, without producing any visible injury, aggravate, by concussion, the "*commotio cerebri*" produced by the violent aerial disturbance. Another and very important factor is "*psychic trauma*," engendered by sudden fear of death as well as by horror caused by the terrible destructive effects on comrades. It has appeared to me that the cases of shell shock have latterly not been so severe as they were in the spring of last year. I attribute this to the fact that life in the trenches has not been associated with such continuous and severe nervous strain and fearful apprehension. Our men have felt that in numbers and in effectiveness of our artillery the balance of power is no longer with the enemy. The joy of hope has replaced the depression of fear.

That "*psychic trauma*" plays a very considerable part in the production of the functional neuroses and psychoses of shell shock without visible injury is shown by the fact that neuro-potentially sound sergeants, non-commissioned officers, and privates, who, after fighting at the front for long periods, have been the subjects of shell shock, as a rule do not manifest severe and prolonged symptoms

of functional neurosis. Officers who have been exposed to shell-fire and have suffered temporary loss of consciousness, according to my experience, recover much more quickly. I have not seen a single case of mutism among the large number of officers that I have had under my care, nor have I, with one exception, seen severe cases of amnesia from shell shock without visible injury in officers; the one case I have seen had a marked retrograde amnesia and chorea, but in his case there was evidence of physical concussion; and this leads me to say that when there is retrograde amnesia there is usually a history of physical concussion, such as the head being struck with a stone or piece of shrapnel, or there is a history of burial. These facts undoubtedly point to the importance of the personal factor. A considerable number of the cases of shell shock without visible injury, in my judgment, occur in individuals of a neuropathic or psychopathic predisposition, or of a timorous or nervous disposition.

As none of the cases of shell shock under my care have died, no opportunity has occurred of examining the central nervous system in the severe cases of shell shock without visible injury admitted to the Fourth London General Hospital.

DISORDERS AND LOSS OF CONSCIOUSNESS.

Shell shock without visible injury is usually followed by a complete loss of consciousness, due to "commotio cerebri" of variable intensity and duration; in some cases the forces generated by the high explosive may cause such a disturbance of functions of the whole central nervous system as to arrest the activities of the vital centres, especially of the medulla, and cause instantaneous death. As I intend to deal with this aspect of the question in the Lettsomian Lectures, which I am giving shortly, I will not touch on this subject now. In the majority of cases the shock mainly affects the cortical structures, the vital centres, as in apoplexy, continuing to function; in some cases the subconscious centres (Jackson's middle level), which preside over the inborn instinctive and habitually acquired purposive sensori-motor actions subserving the preservation of the individual and species, are able to perform their complex functions; but of the experiences the individual has had during this period of automatism he has no recollection whatever. Several cases of this kind have come under my notice, but I will describe only one of the most reliable, as it is a history obtained from an officer. His company had dug themselves in, in a wood; he went out into the road to

iv Mott: *Shell Shock without Visible Signs of Injury*

see if a convoy was coming. A large shell burst near him ; it was about 2 o'clock in the morning, and quite dark ; about 4.30 a.m. it was light, and he found himself being helped off a horse by two women who came out of a farm-house. He had no recollection of anything that happened between the bursting of the shell and this incident. It is interesting to note that the single cigarette in his case was yellow on one side, no doubt due to fumes of the explosive containing picric acid. It is possible, therefore, that noxious fumes may get into the air-passages and be a contributory factor to the physical shock.

Nearly all cases of shell shock are associated with a disturbance or loss of consciousness of variable duration. Sometimes the patient is only dazed, and partially or completely unaware of his surroundings. All degrees of effects on consciousness may be met with, from a slight temporary disturbance to complete unconsciousness with stertorous breathing, continuing till death. It is always, however, difficult to decide from the history obtained from the patient whether the unconsciousness which he speaks of was truly unconsciousness for a whole or a portion of the period, or whether his unconsciousness was not due wholly or partially to the fact that his recollection was blotted out for a time. In support of this I may mention that Major Meyers has informed me that he has been able, by hypnosis, to revive in consciousness events which have happened during this state. Again, it is difficult to decide whether the forces generated by the explosion were alone responsible for the effects on consciousness. Many of those suffering with shell shock without visible injury have been buried, and a sandbag striking the head would easily account for a sudden loss of consciousness ; again, burial or partial burial might be a contributory cause from the production of asphyxia of mechanical origin, or from the enforced inhalation of poisonous gases. The history obtained from comrades and from R.A.M.C. officers at the field hospital was, however, that there may, indeed, be a complete loss of consciousness for days.

Occasionally cases have been admitted under my care who had not yet recovered normal consciousness, and for some days lay in a dazed, somnolent, or even semi-conscious condition. Usually these cases came at a time when large convoys were sent from the Front owing to a recent engagement.

I came to the conclusion, from the investigations made by myself and Dr. Cicely May Peaké,¹ that in the majority of the cases of shell

¹ Dr. Cicely Peaké was for six months research scholar for the Medical Research Committee.

shock resulting in functional paralysis, tremors, tics, and disordered movements; in amaurosis, deafness, and loss or defects of the other special senses; in anaesthesia, hyperaesthesia, and hyperacusis; in speech defects—e.g., mutism, deaf-mutism, aphonia, stammering and stuttering, verbigeration; in amnesias, mental confusion, and other psychic disturbances; in sweats, cold blue hands and feet, and the facial expression of terror or vacant bewilderment; these symptoms, especially if persistent, occurred in individuals who gave a history of a neurotic temperament or neuropathic predisposition. However, in a certain proportion, a little more than one-third, the cumulative effects of stress of active service combined with repeated and prolonged exposure to shell fire or high explosive projectiles apparently had induced a neurasthenic or hysteric condition in the nervous system of a potentially sound individual. I have met with severe cases of shock among good soldiers and non-commissioned officers of years in active service—men of excellent physique who have led active lives, without any evidence of a nervous breakdown. Some of these had served all through the South African War, and others had fought in many actions and battles in this campaign. Such men, as a rule, have not succumbed after a single shell shock, but only after several, and when they have been working under the stress of responsibility, with insufficient sleep and constant apprehension. On the other hand, "among the rapid breakdowns," as a result of shell shock, there are a large number of men who give a history either of a timorous disposition, easily frightened, who feel faint at the sight of blood, or of anxiety neurosis, or of a nervous breakdown in ordinary life, or after some special stress, or of injury to the head causing traumatic neurasthenia. Again a few are mental defectives, as shown by their low wage-earning capacity, their inability to obtain regular employment or to keep their place, or of only having reached a low standard at school. Moreover, their expression is often dull and vacant.

Occasionally cases have been met with where there was a history of the patient having in early life witnessed a terrifying scene, and that this may have produced a deep impression on the mind, predisposing the individual to the emotion of fear, which seems probable from its frequent recurrence in terrifying dreams.

In not a few there was a history of *petit mal* or epilepsy, in spite of the fact that this should have excluded them from the Army as medically unfit. Cases which were said to have developed true epilepsy as a result of shell shock, the history showed, were nearly always individuals who had previously suffered with epilepsy or an anomalous

form of it, or that they were potential epileptics prior to the shock might be assumed from the fact that they had suffered from what they termed "slight faints," or automatisms; or that there was a history of epilepsy or insanity in the family. Some of the cases which showed mental symptoms had been in an asylum, or their parents or some near relative had; these were usually cases in which the mental symptoms which followed the shock were not transitory, but persisted after they had been treated in hospital. These patients not infrequently, prior to the shock, had shown symptoms and signs of mental instability. A few cases developed general paralysis of the insane or syphilitic brain disease, and although shock could not introduce the spirochaetes into the brain, the commotion caused by it might have lowered the resistance and so enabled the latent organism to become active. Although in the large number of cases which have come under my care I have had the opportunity of observing almost all the neurological and mental symptoms which can be produced by shell shock, I shall limit my address to this Section to the more important psychic conditions caused by "commotio cerebri" occurring in shell shock, leaving the neurological symptoms to discussion at the Neurological Section.

Other mental symptoms of "commotio cerebri" from shell shock, especially when there is besides psychic trauma actual concussion, as evidenced by burial and a history of injury or retrograde amnesia, are continued slow reaction, mental dullness, confusion, and inability to undertake any prolonged mental effort without headache and fatigue. There is, however, for some time headache without any exciting cause. Any mental effort requiring sustained attention the subjects of psychasthenia the result of shell shock are unable to undertake, and irresolution and indecision are a frequent result. Officers and non-commissioned officers feel this acutely, and say that they are unable to carry out responsible duties in consequence thereof. This condition is aggravated by insomnia and worry, for they are conscious of their mental state and are filled with apprehension of making mistakes. If there is an inborn insane tendency, this "anxiety neurosis" may be complicated by delusions of persecution and obsessions. In severe cases of "commotio cerebri," especially where there is concussion as well as psychic trauma, the symptoms of Korsakoff's psychosis may occur; thus there may be, as in the cases of severe amnesia narrated, marked mental confusion, disorientation of time and space, mistakes of identity of persons, and especially loss of memory of recent events without delusions or hallucinations.

Hyperacusis is a frequent and very troublesome symptom. The explosion of bombs in a Zeppelin raid had a pronounced effect on many of my cases, causing relapses. The firing of the guns at Woolwich, the noise of passing trains, even of billiards and music, produced in several severe cases distressing symptoms. One case in particular I might cite in illustration. A sergeant reservist who prior to re-enlistment had been a well-known pugilist of great repute, was invalided home for neurasthenia, the result of long active service at the Front. He was progressing favourably when a Zeppelin raid occurred; the noise of the bombs caused a serious relapse. The man was continually making involuntary and uncontrollable movements of the muscles of the face, head and neck, and shoulders, such as he was accustomed to make when fighting. When he was recovering the slightest noise set up these movements. The hyperacusis was so marked that he resented the noise of the billiard balls and would like to have prohibited playing, if necessary by force. The soldiers found the most effective method of causing his retirement was to bang their slippers hard on the ground.

AMNESIA.

Loss of memory is a very frequent result of the effects of high explosive forces upon the central nervous system. It may be a complete loss of power of recollection and of recognition. Consciousness, except for the immediate present perception, may be a blank, and there is in such cases, as a rule, "no art to find the mind's complexion in the face," for a patient so afflicted exhibits a dazed, mindless expression. Three cases in particular I will cite as exhibiting this intense form of amnesia. One case was that of a sapper who, upon admission, could give no information himself, but we learnt from a comrade he had been blown up by the bursting of a large shell in the trench. He could not recollect his own name; he did not recognize his name when it was written; he did not know the season of the year; he could not recognize any coins except a half-crown, but he possessed this coin, which had just been given to him. He did not recollect anything that was told him, so that his memory for recent events as well as for the past was lost. He had a dazed expression, exophthalmos of the right eye, and enlargement of the thyroid, especially of the right lobe. This man rapidly improved and recovered completely.

Another case was remarkable in several ways. The patient's mind was a complete blank, and this condition was reflected in a dazed,

mindless, mask-like expression. When asked where he lived he said "Wakefield"; he did not know it was in the West Riding; he did not know the address of his home, and when shown a letter from his father with the address on the top he did not recognize it, nor his father's handwriting. When shown a photograph of his home with a group of his father, mother, and three brothers and himself in front of it, he maintained the same wondering, dazed expression, and failed to recognize the nature of the picture. His father had heard from a comrade that he had been buried by the explosion of a shell in the trench; he had been unconscious for some time and lost his speech. We heard from his father that he was a good musician, and I said to him, "G., I hear you are a good musician"; so I asked him if he could play the piano or sing. There was the same wondering, bewildered look, and he muttered something which was to the effect that he could not sing or play. Three days later I said, "Come, you can whistle 'God Save the King.' " He took no notice, but upon pressing him he looked up and a glint seemed to come to his eyes as he said, "You start me." I whistled the first bar; he took it up and whistled the tune admirably. I then asked him to whistle "Tipperary," but he could not do this until I started him; and the same with several other tunes. Once started he had no difficulty, and I recognized from the admirable intonation that he was, as his father described him, an excellent musician. I could not, however, that day get him to start upon his own initiative any one of the tunes he had whistled. The next visit, three days later, I observed that his expression had changed. He smiled when I spoke to him, and I recognized clear evidence of a mind that had found itself. He could now whistle any of the tunes I had previously started him on by himself when I called for them. I then said, "Come along to the piano." He came, and I got him to sit down in front of it. I said, "Play." He looked at the instrument with a blank expression as if he had never seen such a thing before, and I could not get him even to put his fingers on the keys. I then took one of his hands, and holding his forefinger, I made him play the melody of "Tipperary." He looked at me, and again I noticed a glint in the eye and a change of his blank expression, indicative of associative and recollective memories. He put his other hand on the keys and played a few chords. I went away feeling confident now that his musical talent would reveal itself. He played for half an hour while I was in the ward without a single discord. The next time I came he was able to play any music set before him. His associative memory and recollection of music were in advance of other associative memories.

Thus eight months after he had recovered his musical memory he had very imperfectly recovered his memory of elementary facts regarding his profession of a land surveyor—e.g., he could not tell me how many poles there were in a rood—and there was still a tendency to a vacant, mindless expression and prolonged reaction-time, as shown by delay and slowness in responding to questions, as if there were a difficulty in linking up the necessary associations.

This early return of the musical memory happened also in another severe case of amnesia which I will briefly relate. This patient was admitted for shell shock. He had almost a complete loss of recollection of all the incidents of his past life except some experiences of early life, such as when he went to school. His powers of recognition were limited to knowing his parents. He had a bewildered, vacant expression and a slow reaction to questions; when interrogated, his countenance assumed the puzzled aspect of one trying to recollect. His memory for recent events was absent, and persons that he had frequently or daily seen he failed to recognize. After four months he had made but little improvement. His memory of the past seemed to show the first signs of awakening in the associations of music. He recollected musicians that he had heard and songs that he had sung, although, as in the case of G., he remembered nothing of his professional occupation. He said that while with his friends he had been asked to sing songs which they said he had sung before—that he did not recognize them at all when he saw them; that after they had been played to him two or three times he was afraid to begin as he felt he did not know them, but that once he started "he seemed to know without remembering" and got through quite well. One song he managed after it had been played through only once ("I hear you calling me").

It may be asked, Why should the memory of music be more readily revived in consciousness than other experiences—for example, those connected with the professions of these two young men before they entered the Army? I would explain it by the fact that there can be no doubt that cognitions, whether pleasurable or painful, are more deeply graven on the mind and more firmly fixed in associative memory when associated with feeling. Music, of all the arts, appeals most to the emotions, and probably this is the reason why countless men and women, even the uneducated, can recall the words of songs and hymns when they hear the first bar of the musical setting.

A short period of amnesia is common in shell shock; in fact, many of the cases of supposed loss of consciousness may really be partially or

wholly due to amnesia. Many instances of high explosions of aerial torpedoes, mines, and large shells, causing shock without visible injury, have occurred resulting in amnesia; but those which have been followed by a complete amnesia or retrograde amnesia have been cases where (as in two of the three first narrated) there has been a history of shock with burial, or there has been visible evidence of concussion, either from the man being blown into the air and coming down on the ground, striking the head, or from a stone hitting his head. In the case of burial, the man may be struck on the head with a sandbag and no visible sign of injury occur, or he may be unconscious in the trench or dug-out, or remain so covered up that he cannot move and thus be exposed to poisonous gases. In respect to concussion producing retrograde amnesia, I will mention one case of many that I have seen; it was that of a young officer who was admitted for acute chorea; there was no history of rheumatism, and I have not any doubt that his condition was due to the shock and concussion. In his case there was a complete retrograde amnesia of two weeks' duration. He could recollect nothing from the time he arrived in Havre till he reached the hospital at Boulogne. The only incident he could vaguely recollect was arms and legs flying into the air, and of this and other terrifying things he dreamed continuously. It turned out that he had been at Hill 60 and the battle of Neuve Chapelle. I asked him to write down any recollections that returned to him. His brother had received a letter from him telling him that he was moving to the Front; he did not recollect it, nor could he tell me anything that happened after arriving at Havre. Reading the newspaper, he saw the word Bailleul, and recalled the fact that this was a place he had been at, but this was merely a word-association, for he had no recollection of the place or of any incident that happened there. I formed the opinion that this retrograde amnesia was due to concussion, how produced we do not know. I asked him to write down for me some of his dreams. They were as follows: playing with red-hot billiard balls after playing billiards; charging up an inclined plane, where a huge Prussian sat down and swept them all away (this dream had doubtless a partial origin in his having seen a torpedo-boat launched); blown up in a house bit by bit; red-hot trenches and burning liquid; in trench by himself. These terrifying dreams continued for a considerable time. The mere bursting of a shell or explosive in the neighbourhood of a man does not appear to do more than produce cerebral commotion and a subsequent amnesia, for, as a rule, the man can recollect everything that happened prior to loss of consciousness. One officer told me as he was blown up

in the air he remembered seeing the terrible effects of the shell as pieces of it struck his comrades.

PSYCHIC TRAUMA AND TERRIFYING DREAMS.

Psychic trauma, in my opinion, plays a very considerable part in the production of symptoms of shell shock without visible injury; in many cases the patient will tell you that he can picture in his mind's eye the shell coming and recollect the sound of the explosion, and even recall its terrifying effects, causing death and destruction of comrades. Seeing that in severe head injuries causing "concussion" there is nearly always some retrograde amnesia, it may be assumed that in these cases "emotional shock" is a very important factor in the resulting "commotio cerebri." The frequency with which these cases of shell shock suffer with terrifying dreams at night and in the half-waking state points to the conclusion that psychic trauma is exercising a powerful influence on the mind by the thoughts reverting to the terrifying experiences they have gone through, and probably their continuous influence on the subconscious mind accounts partially for the terrified or vacant look of depression on the face, the cold blue hands, feeble pulse and respiration, sweats and tremors which some of the severer cases manifest. As these dreams cease to disturb sleep, so these visible physical manifestations of fear pass off and give place to the sweet unconscious quiet of the mind. Occasionally during the waking state contemplation of the horrors seen, visual hallucinations or illusions may lead to motor delirium or insane conduct. At least that is the interpretation I should put upon the symptomatology of the following illustrative cases:—

Case I.—A captain, aged 20, was admitted under my care in a state of restless motor delirium; he moved continually in the bed, sat up, passing his hand across the forehead as if he were witnessing some horrifying sight, and muttering to himself; yet, when interrogated, he answered quite rationally. This motor delirium I associated with the continuous effects on the conscious and subconscious mind of the terrible experiences he had gone through. His whole company had been destroyed, and, while talking to a brother officer, the latter had half his head blown off by a piece of a shell. He improved very much, but a relapse occurred after a night disturbed by terrifying dreams. He recovered sufficiently in a week to go out.

Case II.—*Paroxysmal Attacks of Maniacal Excitement following Shell Shock.*—A young man, aged 19, was admitted suffering from shock, due to emotional stress and shell-fire. He suffered with terrifying dreams, and after

he had been in the hospital a short time he developed sudden paroxysmal attacks of maniacal excitement. The first attack occurred suddenly. One afternoon he had been helping as usual in the kitchen, and then he went and laid down on his bed and apparently went to sleep; he suddenly woke with a startled, terrified look, became flushed in the face, sweated profusely, and made for the door as if to get away from some terrifying conditions. He was with difficulty restrained. He remained in this excited state, glaring rapidly from side to side, giving one the impression that he was suffering from terrifying hallucinations of sight and hearing, although he would make no response to interrogation. He did not recognize his wife, the doctors, or the sisters. Once when I, accompanied by two medical officers in uniform (strangers), came up to speak to him, he became violently agitated, as if some terrifying conditions had been aroused by the sight of the uniforms; the face was flushed and he sweated so profusely that the perspiration dripped in a stream off his nose. The attacks would last from a few hours to a few days; they came on quite suddenly like an epileptic fit, and often without any apparent cause. They became more severe and frequent, and when we had moved the neurasthenic patient to the schools, he one day ran out of the building into the playground and attempted to get over the wall. He was brought back, and I saw him sitting in the ward on his bed; his head was buried in his hands. I spoke to him; he immediately got up, looked at me in the most terrified manner, and made for the door; it required four orderlies to restrain him, and he fought and kicked violently, exhibiting great strength and nervous energy. Much to my regret, I found it necessary to have him sent to Napsbury. I have heard that he has made a complete recovery and been discharged. It may be mentioned that there was no history of epilepsy or insanity in the family obtainable. The case rather suggests the psychic equivalents of epilepsy in the attacks.

Case III.—Traumatic Neurasthenia prior to Enlistment; Emotional Shock.—Private P., aged 25, admitted on November 11, 1915; occupation, a coal-miner. He had a bicycle accident five years ago, was unconscious two and a half hours, off work for five weeks; ever since has had headaches and been subject to fainting fits and nervousness. He has imagined "he saw things when there was nothing to be seen." On September 19 he was under heavy shell-fire in trench and dug-out. He saw the sergeant and three men who had been working with him killed by a big explosion, and remembers his cap being lifted off his head. He came to in 46 Rest Camp; he does not know how long afterwards. He could not see clearly and could not hear or speak; he had bad headache and could not sleep. The notes which accompanied him from the hospital in France related that he was deaf and dumb from shock, but that he could write intelligently. The paper in his handwriting makes this statement: "Doctor! I had an awful dream last night again; I was dreaming that I was in the trenches. I could see the men falling and the great big shells exploding. I could see the light from the bursting of the shells very plain; they fairly lighted all the place up. I woke up very

nervous, I can tell you. I wish that I could give over dreaming, and I keep having pains in my head right across my eyes." He recovered his speech in the following manner: On October 15, while sitting outside by himself, he felt a slight crackling in his head, and then noticed he could hear sounds faintly, and in a few minutes he could hear fairly well. On October 17 he was heard making inarticulate noises in his sleep. The corporal next him told him he had been making noises, and in a half-drowsy state he tried to speak. He said "Mother," then felt "very queer all over, with pain in his head." After this he was able to talk very well with only very slight hesitation.

Case IV.—Shell Shock and Psychic Trauma from witnessing Death of Comrades; Psychic Trauma maintained by Terrifying Experiences and Dreams; Nervous Predisposition; Recovery after Six Months.—Private L. R., aged 21. Admitted to Fourth London General Hospital on June 20, 1915. Was at Boulogne about two weeks. Present condition: Lying in bed on back; continuous jerky lateral movements of head and movements of the arms, especially the left. He utters slight groans, and every now and then the eyelids are elevated, producing a staring expression and look of bewilderment and terror. Mouth is slightly open. He appears to comprehend what is said to him and answers questions in a muttered whisper. He occasionally raises his right hand to his forehead. The movements are exaggerated when he is being observed. The movements cease during sleep and become much less when he thinks he is not observed, but the muttering continues. He continually says: "You won't let me back." Asked about what he dreams he says: "Guns." The pulse is fairly good; no cold sweats; hands and feet not blue. On attempting to obtain reflexes he exerts voluntary movements which prevent their being obtained. No Babinski sign. The jerky movements of the head continue even while he drinks. When I went to look at the pupils they dilated, and there was a marked facies of terror. The eyes were opened wide and staring, transverse furrows were formed in the brow, and the inner extremities of the eyebrows were contracted by the corrugators. Apparently this was from the impression caused by seeing a uniform. (He has a continued dread of being sent back.) A marked similar effect was produced by the flash of an electric lamp under his eyes. June 24: Much better. Spoken words more intelligible. He had only been a few weeks at the Front when "knocked out." It occurred in this way: He was carrying sandbags with thirty men in, daylight under shell-fire. One explosion flung him into a deep hole, and he climbed out (no loss of consciousness) to see all his friends lying dead around him. This was his first sight of any bad injury, and he keeps seeing it again with bright lights and bursting shells. He does not hear the shells, but sometimes the men shouting. He dreams he sees and hears both shells and men. Complains of pain in back and right side of head. June 26: Improved. No involuntary movements except slight tremor of hands. Still pain in back of head, especially when he tries to remember. He remembers being very cold and shivering in hospital at Boulogne, and being given many hot baths. Does

not know the day or date, but knows it is morning (11 a.m.). Says he has always felt sick at the sight of blood, and that he had very bad headaches from the sun when he first went out (May, 1915). No cranial nerve paralysis. Pupils equal, slightly dilated, react well to light, less to accommodation (still reads even large print with great difficulty). Anæsthetic to pain and light; touch arms and trunk. Knee-jerks, abdominal reflexes, and ankle-jerk very brisk. Flexor plantar response. Dreams he sees and hears shells bursting. Sent to Morden Hall six months later, boarded for home service.

THE ORIGIN OF DREAMS.

Dreams are not "born of fantasy, children of an idle brain," but are based upon experiences of the past, often linked up in incongruous association. I could cite many instances of this: two will suffice. An officer who had served in South Africa told me that he had had a dream from which he awoke in a fright. He was in a mine passage at the Front when he met a leper, who came towards him. Upon questioning him and asking him to recall some period of his life in which his mind had been disturbed by a leper, he said that when serving in South Africa he remembered that he and his comrades were very much upset by a leper being allowed to remain in an adjoining sangar.

A sergeant who had been a school-master was asked to write down his dreams: "I appeared to be resting on the roadside when a woman (unknown) called me to see her husband's (a comrade) body, which was about to be buried. I went to a field in which was a pit, and near the edge four or five dead bodies. In a hand-cart near by was a legless body, the head of which was hidden from sight by a slab of stone. (He had seen a legless body, which was covered with a mackintosh sheet, which he removed.) On moving the stone I found the body alive, and the head spoke to me, imploring me to see that it was not buried. Burial party arrived, and I was myself about to be buried with the legless body when I awoke."

Terror is contemplative fear, it is fear made more or less permanent by the imagination fixing in the memory past terrifying experiences, repressed in great measure by conscious activity of the mind during the waking state, but evident in the dreams which afflict nearly all these soldiers suffering from shell shock and trench warfare. Shakespeare has not only in his characters shown how a passion steals into the soul, so that it becomes the sole tyrant of the desires, but he has clearly indicated how dreams influence the minds of men, and how they are based upon past experiences. Thus Mercutio, in the description of

Queen Mab, refers to the soldiers' dreams in the following lines, which are as true to-day as when Shakespeare wrote them :—

Sometimes she driveth o'er a soldier's neck,
And then dreams he of cutting foreign throats,
Of breaches, ambuscadoes, Spanish blades,
Of healths five fathom deep ; and then anon
Drums in his ear ; at which he starts and wakes ;
And, being thus frightened, swears a prayer or two,
And sleeps again.

Although this terror has a more profound influence upon a neuropathic individual, yet all the symptoms I have mentioned may be induced in an individual of sound neuropotentiality, but as in the case of the captain, recovery is more likely to occur.

The following case is of interest in connexion with dreams of past experiences :—

A deaf-mute, aged 24, with no history of a neurotic temperament or neuropathic predisposition, was admitted under my care on November 16, 1915. He wrote the following account of himself : " I left England the 8th March and went to Gallipoli on the 26th May, and about the middle of August one of our monitors fired short. I felt something go in my head, then I went to the Canada Hospital ; they said it was concussion." In answer to questions he says the last thing he remembers is seeing the monitors firing. He came to in a dug-out about one hour after. He could see and speak a little, but was quite deaf, and his head felt as if it would burst. He lost his speech completely when Bárány's test (hot and cold water tests) were applied. He does not now complain of headache, but is quite deaf and dumb. Captain Jenkins reports that the ears are normal ; it is therefore a functional deafness. He is able to cough and whistle, but cannot speak. His wife says that she has letters from him, in one of which he described how he killed a Turkish woman sniper. He does not remember writing this letter, but there is evidently some retrograde amnesia. He says he does not dream, but it seems certain that he has dreams but does not recollect them, for the sister of the ward says that while asleep he assumes the attitude of shooting with his rifle, and he gives a jerk as if pulling the trigger, then he assumes the attitude of using his bayonet ; the other men in the ward tell her that he gives the movement of the right parry, then the left parry, and lastly the thrust, as if he were in action. He sometimes jumps his whole body as if he heard or saw a shell coming, and he catches his right elbow as if he were hit there. He was then observed to open his eyes wide, get up, and look under the bed. Apparently he is not conscious of this. He then awakens and begins to cry, but there is no sound. These defensive habitual attitudes have been also noticed in soldiers when under an anæsthetic. During hypnotic sleep he did not assume these defensive attitudes, although he dreamt of his trench experiences.

SPEECH DEFECTS.

✓ About one in twenty of those suffering with shell shock and having no visible signs of injury have lost their speech, and yet are quite able on admission to write a lucid account of their experiences. As a rule, these men are unable to whisper or produce any audible sound; thus there is no sound when they laugh. They are unable to whistle or to cough, and in severe cases there is difficulty of putting out the tongue, and in one case of swallowing. Why should these men whose silent thoughts are perfect be unable to speak? They comprehend all that is said to them unless they are deaf, but it is quite clear that in these cases their internal language is unaffected, for they are able to express their thoughts and judgments perfectly well by writing, even if they are deaf. The mutism is therefore not due to an intellectual defect, nor is it due to volitional inhibition of language in silent thought. Hearing—the primary incitation to vocalization and speech—is usually unaffected, yet they are unable to speak; they cannot even whisper, cough, whistle, or laugh aloud. Many who are unable to speak voluntarily yet call out in their dreams expressions they have used in trench warfare and battle. Sometimes this is followed by return of speech, but more often not. One man continually shouted out in his sleep, but he did not recover voluntary speech till enjoying the festivities of singing the old year out and the new year in, eight months after he had suffered the shock. It was thought that his loss of speech might be connected with adenoids, and he was told that if these were removed under an anæsthetic he would certainly be cured; but in spite of the strong suggestion he did not recover his speech. In another case of a severe character the larynx was faradized, but it made no difference; in fact, it did harm, as it increased the emotional condition. The sudden and varied manner in which these mutes recover their power of articulate speech and phonation is indicative of a refractory condition of the voluntary cortical mechanism of phonation. In some cases there is a history of a blow on the chest—e.g., from a sandbag—or of being buried and partly asphyxiated; and it is usual for the loss of speech to occur at the time of the shock. One patient, however, gave a history of difficult speech for two days after the shock—he lost his speech completely only after his vestibular reactions had been tested; while another who, after the same investigation, became a deaf-mute recovered his speech upon hearing a man in the hospital say the word “Rose”; he at once sat up and repeated the word, proving, as he said, “I could both speak and hear.”

Some of the earlier and more severe cases of shock followed by mutism were unable to expire forcibly enough to cough, whistle, or blow out a candle, but the less severe may be able to perform these acts and yet be unable to speak or whisper. The latter cases recover usually more quickly than the former, but sudden recovery may occur in the severe cases. Thus a private, who went to France in October, 1914, on August 9, 1915, was going to pick up a wounded comrade when a shell came and blew his comrade to pieces, and he then knew no more till about half an hour later, when he recovered consciousness, but found himself deaf and dumb. There evidently were two factors in the production of the symptoms: the physical and the psychical. He was admitted under my care. Some weeks later his fellow-soldiers thought he ought to hear and speak, and they adopted energetic measures to make him shout out for help. Two of them leathered him with a slipper and then nearly throttled him. He struggled and shouted "Stop it." Another man dreamt he was falling over a cliff, shouted out, and recovered his speech; another dreamt he was blown up by a trench mortar, and shouted for help; finding himself speaking, he continued to speak aloud, and did not go to sleep again for fear he might lose his speech. Another man, a deaf-mute, was heard to speak in his sleep. He was told by a comrade. He said, "I don't believe it." Some have suddenly recovered their speech by crying out when unexpectedly feeling physical or mental pain; for example, one man cried out when some boiling tea was spilt over him, another when he was held down and his feet tickled. In most cases it is the sudden and unexpected which restores the function of the vocal mechanism. Thus a mute sergeant saw some soldiers larking in a punt, and he suddenly shouted out, "You will be over." Occasionally the stimulus of a well-known chorus has broken down the refractory condition in the psychic mechanism of the voice, and the mute has surprised himself and others by finding himself singing. This mutism is due to an emotional shock; it is a psychic rather than physical trauma in my judgment, for it in no way differs from the description of hysterical mutism. "Some of the leading peculiarities of hysterical mutism are these: its onset is very sudden, and often after a fright or some strong emotional disturbance; sometimes it follows an hysterical seizure, either with or without paralysis of limbs; at other times it occurs without assignable cause, or it may be induced, as already stated, in some hypnotized persons by suggestion. The subjects of this disability are completely mute—presenting in this latter respect a notable contrast to ordinary aphasics, who so frequently make use of recurring

utterances or articulate sounds of some kind. The intellect seems unimpaired, and they are able freely to express their thoughts by writing. Though the common movements of the lips, tongue, and palate are preserved, these parts (constituting the oral mechanism) are unable to act in the particular combinations needful for speech movements, in association with the other combinations of muscular action pertaining to the vocal mechanism.

Bastian¹ notes also that there may be more or less complete anaesthesia of the pharynx in hysterical mutism; this I have observed in some of the mutes. He notes that, as in these soldier mutes, hysterics may recover their speech suddenly as a result of a strong emotion; also, as in the soldiers, recovery may be followed by stammering or stuttering. I have noticed that the stammering more frequently occurs in a person who had a tendency to stammer before the shock occurred.

Bastian refers to a case in which frequently recurring attacks of deafness were generally associated with blindness or deafness, one or both. These conditions are also observed associated with mutism in soldiers the subjects of shell shock. He also cites a case of his own—a sailor who suffered with a great number of attacks of mutism (the first occurring as a result of fright) who previously had not suffered from any nervous disease and previously had led an active life in all parts of the world. We may therefore conclude that this mutism resulting after shell shock in no way differs from hysterical mutism. It appears, therefore, that there is nothing new in these functional disturbances and disabilities of speech and special senses except it be their severity and frequency in men the subject of shell shock.

We may now inquire into the pathogenesis of mutism. Charcot attempted to draw a distinction between aphonia and mutism. He adopted the doctrine of Marey and other physiologists that the larynx takes no part in whispered sounds. According to Charcot, therefore, aphonia (in which the power of whispering is preserved) is a result of a partial paralysis of the adductor muscles of the larynx; while as to hysterical mutism, Charcot writes: "If the individual suffering from the affection is unable to whisper, it is not because he is aphonic, or rather because his vocal cords do not vibrate; it is not because he has lost the common movements of tongue and lips—you have seen that this patient was able to blow and whistle; it is because he lacks the ability to execute the proper specialized movements necessary for the articulation of words. In other terms, he is deprived of the motor

¹ Bastian, "Aphasia and other Speech Defects," p. 126.

representations necessary for the calling into play of articulate speech." Charcot therefore believed the oral division of the speech mechanism only to be at fault in hysterical mutism. Wyllie maintains that whilst this may be so in some cases, in a second group it is the laryngeal division of the speech mechanism which is at fault, and in a third set of cases both oral and laryngeal mechanisms are simultaneously disabled. Bastian alludes to a remarkable case recorded by Guido Banti, in which the individual lost his speech owing to a lesion of Broca's convolution, but was able to write and the general intelligence was not interfered with. Charcot considered hysterical mutism to be an instance of pure "motor aphasia," resulting from a functional trouble in Broca's region. Bastian, however, agrees with Wyllie that aphonia and mutism are most intimately related, differing in degrees only; "and the oral and vocal speech mechanisms are concerned in all speech mechanisms, whether it is sonorous or whispered." Bastian considers that the clinical differences between simple aphasia and hysterical mutism force us to believe in the existence of a bilateral cortical disability in the third inferior frontal convolution.

Sir Charles Bell, in his great work on the expression of the emotions, first drew attention to the influence which powerful emotions exercise upon the respiration. A part of the cortex controls the mechanism of breathing in the production of all voluntary audible sounds, and this, like the movements of the vocal cords, is represented in both halves of the brain, for the muscles of the two sides of the body which control the breath and phonation always act synergically and never work independently. In the oral division of speech mechanism the muscles of one side never act independently of the other. Bastian is probably therefore correct in asserting that it is a functional disability of cortical structures in both hemispheres. Whether he is right in asserting that it may be localized in the third inferior frontal is another matter. I believe this mutism is due to an inhibitory functional paralysis of the voluntary central nervous centres which control the management of the breath and its mode of escape through the glottis, mouth, and nostrils, for I have seen many cases where they have involuntarily and unconsciously in their dreams talked and uttered cries and swears, but in their conscious state were unable not only to talk or sing, but to whisper, whistle, utter a cry, cough, or laugh aloud. The worst cases were unable even to blow out a candle. I examined one with X-rays and found the diaphragm could by no effort of the will be made to descend in an adequate way to fill the bellows sufficiently to produce an expiratory blast for coughing.

The sudden mode in which these mutes recover is remarkable, some trivial circumstance bringing it about—e.g., a mute went out of the ward and to his surprise met an orderly, who asked him what he was doing there; he replied he did not know he was doing wrong. The element of surprise and unconscious effort seems to be essential. The more they wish to recover the less likely they are to do it. Conscious efforts, therefore, will not help. I tried offering a bottle of stout to a man as soon as he spoke; it had no effect. This is, however, a useful method to apply to soldiers who have been mute and who are not desirous of getting well too soon. One man I had who screwed up his face as if he could only articulate in a whisper with the greatest efforts of the will. He over-acted the part. "Sister," I said, "this patient is ill: he should be kept in bed and put on No. 1 diet. When he asks loud enough for you to hear he may have a mutton chop and a bottle of stout." He was not long in recovering! A case which did not recover for eight months was most anxious to recover and tried very hard; he became depressed when he saw others had recovered their speech. All these facts make it probable that an *anxiety neurosis* keeps up this mutism by dissociating the cortical ideation neurones of internal language and feeling from the effector neurones which direct and control the breath and its mode of escape. Why should they be able to write and yet not produce audible sounds? Written language expresses comparatively little feeling as compared with spoken language in which, by the control of the breath and phonation, the voice is modulated and suffused with the appropriate affective tone to express the various emotions and passions. Moreover, inarticulate audible sounds, expressive of the primitive emotions and passions dependent upon the management of the breath and phonation, constitute an important part of a universal language understood by all mankind. The mutism following shell shock cannot be explained by cerebral commotion caused by the dynamic force generated by the explosive in a definite anatomical region of the brain, but must be associated with emotional shock caused by terror, which would affect the whole seat of consciousness.

SUGGESTION.

Suggestion plays an important part in the determination of functional disabilities. Thus one patient was blown up and fell on his right side, bruising the right leg: he afterwards had a functional paralysis of that limb. Some of the cases of mutism followed burial and compression of the chest. A soldier became a functional deaf-mute after the Bárány

test had been applied (*vide p. xv*). A case of functional amaurosis was interesting: a shell burst near a man and wounded a comrade; he dragged the wounded man into a culvert; there he lost his sight, and he could not have found his way out if another wounded man who had taken refuge there had not assisted him. He could see only dimly, as through ground glass, when admitted; he could not distinguish colours or the form of objects at first. He recovered quite suddenly after I had paid a visit and tested him. It is possible that in this case the darkness of the culvert had suggested the loss of vision, and set up a dissociation between the cortical visual perceptor and the terminals of the visual path to the cortex.

Case to illustrate the influence of personal suggestion of recovery by the abolition of a fixed idea.

Shell Shock, without Visible Injury, from a 17-in. gun, affecting a Gunner of Fifteen Years' Service; Severe and Continuous Symptoms following Loss of Consciousness; Tremors, Analgesia and other Sensory Disturbances, Hyperacusis.—Gunner H. A. S., aged 39; fifteen years' service R.F.A., 49th Battery. Out in France ten months; admitted Fourth London General Hospital in January, 1916. Last August, between Ypres and Flamentières, about 3 o'clock in the morning, a Jack Johnson exploded near him, and he remembered no more until he found himself in the Military Hospital, Chatham, fourteen days later. The Colonel told him he was lucky to be there at all. He believes the shell killed a number of his comrades. He was afterwards transferred to Colchester, and from there to the Fourth London General Hospital. When I examined him he was sitting in a chair; legs, hands, and jaw were in continuous rhythmical tremor, exaggerated when he was spoken to; the tremor resembled a clonic spasm so large was the amplitude of the movement. Every now and again he starts, and looks laterally and upwards as if he feared a shell would drop on him; his hearing is extremely acute—in fact, there is hyperacusis—the firing of the guns at Woolwich causes him alarm. In narrating his history there is a constant repetition of words—e.g., he said "in action" five times, "between" five times, and then finished the sentence. He dreams of shells bursting, and a fellow-patient says he has disturbed sleep, groans, and utters moans and sounds, and he wakes up with a start at the least noise. It is very difficult to test the reflexes on account of the continuous coarse tremor. The face is flushed, the palms sweating, but the surface temperature is not lower than normal. The pulse is good. He states that he feels better, he can now feel the floor, but he cannot stand or walk without assistance, largely because there is constant tremor. Sensibility and special senses: He does not feel the prick of a needle on the lower limbs, or left arm and hand, but responds on right hand after delay. On the face he responded immediately. He does not feel the vibrations of the tuning-fork on the feet, legs, or hands, but does on forehead, and there was a marked contrast between the bone sensibility of

these parts. He hears the fork quite well at 6 in. from the ears. He has some difficulty in recognizing colours: he picked up the blue box, but was a long time before he picked up the red. His sense of weight was not abolished, but it was difficult to test because of the tremor, which became of much greater amplitude, resembling, in fact, the intentional tremor of disseminated sclerosis, causing the pennies to rattle in the boxes. It is possible that his judgment of the relative weights was aided by hearing the pennies rattle. He could taste very bitter fluids, but could not recognize vinegar, salt, or sapid fluids. The sense of smell was considerably affected, for he did not recognize tincture of asafoetida, attar of roses, and oil of cloves; he smelt nitrite of amyl, and recognized strong ammonia and glacial acetic acid, but they seemed to have much less stimulating effect than might have been expected. The long time which has elapsed since the shell shock, and the severity of the symptoms which have persisted, coupled with the fact that the man was a gunner of fifteen years' service, renders it probable that this may be a case in which functional disturbance due to emotional shock is not the sole cause of the symptoms.

Although I felt that there was possibly some organic change in his brain to account for the severity and persistence of these rhythmical tremors, increased by attention, yet I felt sure that the only hope of recovery was by suggestion; therefore I assured this man that he would recover and that the tremors would cease. I told him that the careful examination I had made assured me that there was no organic disease and made me certain that he would recover. A steady improvement has occurred, and yesterday, a fortnight after admission, he is sitting in his chair without any tremors and with a profound belief in me, and most grateful. I use him now as an object-lesson to all those who are affected with tremors. If I had used hypnotism I do not think I could have done better, and I might have done worse. Captain Brown, who looks after my cases, will tell you of a case where the arousing during hypnotic sleep of terrifying experiences in dreams had a very bad effect on one of my patients. However, I do not think this should stop us using hypnotism in cases where suggestion by personal influence fails. I will relate another case, one of paraplegia, which I saw at a convalescent home, that was supposed to be of organic origin and had been massaged and electrified for months. I examined him and found no signs of organic disease. I said: "Get up"; "I can't," he said. "I will help you," I replied, and at first I supported him, then, while conversing with him, I relaxed the hold I had, and after a little while I told him that he had been standing by himself, and if he could stand he could walk, which I made him do, supporting him a little. "Sister," I said, "take away that wheeled chair, take away sticks. He must get up

and walk without any aid." A month later when I visited I found him quite well. I could relate experiences of malingerers and the difficulties experienced in discovering them, but possibly some cases which one suspects could only be found out in a way which was suggested to me in a dream. A man that I felt convinced was a malingerer, but I could not decide whether he might not be a case of astasia-abasia, was boarded out of the Service. I dreamt about this man being before a medical board, and that he was made to walk by the threat that if he did not do so he would be court-martialled and shot the next morning.

I will conclude by briefly summarizing some of the remaining important functional disturbances noted in these cases of shell shock.

Hearing.—There may be complete deafness as a result of the shock; in some cases sounds are heard, but their meaning is not comprehended. In many cases there is at first an organic cause for the deafness due to the explosion; this deafness is worse on the side on which the shell has burst. In some cases the drum is found to have been damped off by wax which has been forcibly driven against it. Hyperacusis is a common and a very troublesome symptom, causing apprehension and even morbid fear of noises. Hallucinations of hearing are fairly common—the patients complain of hearing shells coming, of bullets whizzing and whistles blowing. Many get a drumming in their ears, especially when they have had headache, but this may be replaced by ticking or clucking.

Vision.—This also may be completely disturbed during the acute stage of shell shock, but more often the patient complains of "smoky vision" than blindness. Attacks of smoky vision may occur. Failure of accommodation and feeble reaction to light are common in the acute stages, and it may be weeks before the patient is able to read even the largest print. Occasionally there is diminution of the colour sense and of the visual field. One officer, a Canadian, complained of seeing everything greatly magnified—his bandolier of cartridges looked as large as a pom-pom belt.

Illusions and hallucinations are not uncommon symptoms in the acute stage of shell shock.

Headache invariably follows shell shock: it affects especially the occipital region, passing down the back of the neck; other positions are frontal, supra-orbital and vertical, in one or both temples, or behind the eyes. The pain is variously described as burning, stabbing, like a tight-fitting helmet or hat, or like a hot wire being run through the head.

At the same time there may be a dull aching. It is worse at night, especially on lying down and on trying to go to sleep. It seems often to be associated with the revival of terrifying thoughts, and is increased by the mind dwelling on them and trying to repress them. Some of the mutes complain of headache when they make efforts to speak, and deaf men when they make efforts to hear, or the functionally paralysed to use his limbs.

Headache.—Conscious effort—attention—uses up natural energy, and this gives rise to headache. Strange to say, some few patients on suddenly recovering their speech have felt ill and complained of headache for some hours afterwards. Headache is aggravated by noise, and insomnia and sleep disturbed by terrific dreams, and by physical or mental effort; it is one of the most intractable symptoms of shell shock. It only passes off gradually, and may be so severe as to give rise to depression, melancholy delusions and suicidal tendencies. Inability to concentrate attention and irresolution are common symptoms.

I will not do more than mention the fact that the motor disturbances may be various—tremors, tics and paralyses. These are:—

(1) General tremors, which may be very marked, especially in the acute stage, coarse and medium.

(2) The tremors, which may be coarse, medium, or fine, limited to the hands. There may be tremors of the tongue and face muscles, like those seen in general paralysis of the insane.

(3) The tremors affecting the legs are coarse and exaggerated by attempts to move them, especially if the patient attempts to walk.

Various tics are observed. Torticollis, facial spasm, blepharospasm and lateral movements of the head upwards and over the shoulder, as if anticipating a shell coming.

Gaits.—A curious dancing gait, a short rapid step, hardly raising the feet from the ground, a shuffling of feet along the ground, making hardly any progress. Inability to stand or walk. Astasia-abasia.

I trust that our neurological colleagues will fill up the many gaps I have purposely left; for to deal with such a vast subject is more than can be expected. I have therefore dwelt especially upon some of the more interesting psychical aspects of the subject with a view to promote, at this Section, a discussion thereon.

In the Lettsomian Lectures which I shall deliver at the Medical Society I hope to have time to deal more fully with the subject of the possibility of the causes underlying "commotio cerebri" following shell shock.

Major W. McDougall, R.A.M.C.

At Netley, where I have been in charge of the Neurological Section since last May, the shock cases are of especial interest to members of this Section of the Royal Society of Medicine, for all Expeditionary cases labelled "mental" are sent there in the first instance. That is to say that at Netley we see many shock cases which present positive symptoms of mental disease in addition to, or in place of, the functional disabilities of mind and body which are the most frequent effects of shell shock. We see, therefore, many cases of mental disorder in early or incipient stages. In a surprisingly large number of cases the symptoms are sufficiently pronounced to lead the officers who see them abroad to the diagnosis of definite mental disease. The diseases most frequently diagnosed (often by officers of asylum experience) seem to be dementia præcox, melancholia, paranoia, mania, and stupor. The following points of interest occur to my mind in connexion with these cases. In a large proportion of them the symptoms clear up very rapidly. In explanation of this fact I would suggest the view that in these rapidly recovering cases we are dealing with persons whose constitutions are naturally stable and elastic—constitutions, that is to say, which were liable to break down only under the extremely severe shocks and stresses of modern warfare. Another point of interest is the fact that the leading mental symptoms, such as delusions and hallucinations, are so frequently traceable to particular experiences of a vivid and generally of a distressing kind.

As the period of the War lengthens out, I am increasingly impressed by the frequency of cases of the following character: A man sustains shell shock, and is sent home with some functional disability such as amnesia, mutism, paralysis, or tremor. After some months he returns to the Front seemingly cured, and there he very soon develops positive symptoms of mental disease. Another way in which the profoundly disturbing nature of shell shock reveals itself is illustrated by cases of the following kind: A man passes many months at the Front, sustaining with equanimity the various stresses, until one day a shell explodes near him without incapacitating him from duty, perhaps merely causing him to be shaky and dazed for a few minutes. From that time on he is a changed man; he can no longer come under shell fire without distress; he is always anticipating the shells with dread, and before long he breaks down with hallucinations or delusions or all the symptoms of mania or of melancholia.

My experience leads me to agree with Dr. Mott's observation that at present functional disabilities due to shell shock are less frequent and profound than they were earlier in the War; and I am inclined to accept the explanation which he has suggested this evening. If this view is true, we may see in it one among many other indications that shell shock is in many or, as I think, in almost all cases, not a merely physical concussion, but involves, as an element of large and often preponderating importance, a moral or emotional shock.

As I listened to Dr. Mott's comprehensive and most interesting account, I tried to call to mind cases of types not described by him, and I have found only one such. This is a type of which I have seen only two well-marked instances. It may be roughly described by saying that the patient is reduced, both intellectually and morally, to childishness. One of these cases has been discharged recovered, the other remains very obstinate.

Very interesting and also satisfactory (in that they are readily curable by suggestion) are the cases of simple amnesia following on shell shock. I am disposed to question Dr. Mott's generalization that retrograde amnesia results only from physical concussion and not from the mere explosion of a shell in the vicinity of the patient. For I have had under my care several cases of very profound retrograde amnesia in which there has been no bruise or other evidence of concussion by impact with a solid body. One of these cases was especially interesting in that the patient's memory function seemed to be reduced to its most rudimentary or primitive condition, such as we may suppose to obtain among the lower animals. It was eventually proved that he had been a barber; and we were led to suspect this by the fact that, when a pair of scissors was put into his hand and he was asked to cut the hair of another patient, he showed great aptitude for the task and said it seemed familiar. When an old friend entered his ward for the first time, he started up, calling out the Christian name of the visitor; yet he could recall no further fact connected with him and did not know that he had seen him before. Again, although the patient had been a choir-boy, he could not recall any melodies, and when a popular air was whistled before him, he did not recognize it; but he reproduced it accurately and said that as he whistled the air it seemed familiar.

But the lateness of the hour forbids me to pursue further any of the multitude of interesting questions raised by Dr. Mott.

Captain WILLIAM BROWN, R.A.M.C., M.B.

Speaking as a psychologist rather than as a neurologist, I should like to mention quite briefly a few facts that I have been able to observe while treating shell shock cases at Maghull and at the Fourth London General Hospital. At the latter hospital I have had the privilege of working under the direction of Dr. Mott, and have treated two of the cases to which he refers in his paper. The deaf and dumb patient from Gallipoli was hypnotized by me repeatedly. In hypnosis, somewhat to my surprise, he did not go through the movements of fighting, &c., which often occurred during his ordinary sleep, but on being brought out of the hypnotic state he intimated by writing that he had been dreaming that he was back in Gallipoli. I hypnotized him repeatedly, and after each hypnosis he described some part of his experiences in Gallipoli, the accounts when put together being a fairly complete record of his terrifying experiences while on the Peninsula. But he is still unable either to hear or speak. I have not completely given up hope, but the conditions are difficult, since no suggestion can be given to the patient during hypnosis. I mention the case to show that, in one instance at any rate, mere hypnosis in itself is not sufficient to effect a cure. I have tried hypnosis with a number of other cases, and to my own surprise did not meet with a single failure. But the effects of the hypnosis have not always been good. One patient, with a tremor of the left hand, found himself back in France under shell fire during hypnosis, and despite suggestions made to him in this condition, his arm trembled more than ever, and on being brought out of the trance he implored me not to repeat the treatment, as the events he had been re-experiencing were too awful. He had just seen his "pal's" head blown from his body by a shell. When the lost memories are not too painful, a patient may undoubtedly benefit by their recall through hypnosis.

With regard to the dream which Dr. Mott quoted, in which a legless body figured, I might add a few remarks. The patient who dreamt this dream is a well-educated sergeant, and by means of mental analysis—I will not call it psycho-analysis—I have discovered that all his dreams are full of significance, and his other mental symptoms likewise. He dreams repeatedly of a wounded man taken up on the field of battle by one of his stretcher-bearers, who had had both of his legs shot away; but in the dream the head of the wounded man is replaced by that of the patient's wife or mother. Anxiety about his wife, whom he had recently married and who was much younger than himself, was found

to explain this characteristic of the dream. Thoughts about his mother were involved in the same worry. The patient had suffered from several fainting fits since falling ill in France, and himself remarked on the fact that they always occurred in the dark. While I was cross-questioning him about this he suddenly remembered that he had received a very severe fright one dark night near his dug-out. He went out to view some dead bodies, one of which was the body of a close friend. On drawing aside the macintosh sheet from this body, to his horror it stretched out its arms, although its head was smashed in and it was otherwise severely mutilated. In fact, the man was alive and did not die till four hours later.

The patient himself volunteered the suggestion that this was the origin of his fits at night. The incident appears, with very little alteration, in the dream which Dr. Mott has quoted. This same patient tells me that he now notices a tendency in himself to become suspicious of people around him on slight provocation. In particular, he feels irrationally suspicious of his wife, without any justification whatever. Cross-questioning elicited the fact that the patient, when about the same age that his wife now is, had become somewhat intimate with a married woman, and he now admits that he subconsciously fears lest the tables may be turned upon himself. In ways like this it has been possible to show and—what is most important—to convince the patient himself that his symptoms, apparently so irrational, are adequately explicable in terms of his earlier history. The effect of this mental analysis on his mind seems to be definitely beneficial.

This case brings me to a brief illustration of the kind of work which I have been doing at Maghull, under Major R. G. Rows. Major Rows, with his splendid enthusiasm for the method of analysis and re-education, has encouraged the same interest in his staff, and has given every opportunity and facility for detailed work upon these lines. Out of numerous analyses which I have of my own cases, some extending over many months, I will mention two very briefly, as illustrations of the method and its *rationale*.

One is the case of a young soldier suffering from hallucinations of hearing. He was continually hearing the voices of his brother and sister upbraiding or admonishing him. He called it "supervision," and his own theory was that it was a case of telepathy, and that the supervision was intentional. Investigation showed that he first began to hear these voices five years previously, three months after returning to his parents from a holiday at Brighton, where for the first and only time in his life he had gone with a woman off the street. In the train

on his way home he became physically sick, for no reason whatever so far as he could discover. It became quite evident on analysis that this yielding to temptation on the part of a well-educated son of respectable parents had been the chief factor in the origination of the disease. The vomiting was a translation into the physical of the subconscious mental, and the voices heard later were the externalization of the boy's feelings of remorse and lack of sureness of himself. There were many additional symptoms and factors which I cannot go into here. My treatment consisted in revealing to the patient, through his own confessions, the chain of mental causation which led up to his symptoms, and in persuading him to accept this view in the place of the systematized delusions of telepathy and supervision which he had been fostering. As my aim was to give him self-knowledge, I would like to call the method which I used that of *autognosis*. The patient made a complete recovery, and was discharged within three months. Of course there is the possible objection to this, as to every other case of recovery from mental trouble, that rest and mere lapse of time were the efficient factors leading to recovery, and that my argument is an instance of *post hoc ergo propter hoc*. To this I would reply that it *may* be so, but that actual experience of the individual cases as one analyses them gives rise to a compelling feeling of causal connexion. There is always collateral evidence of a confirmatory nature which helps to pin one down to a single hypothesis.

My other example is that of a sergeant who was in the firing line during the retirement from Mons. He was admitted to Maghull suffering from the delusion that people thought he had himself given the signal for the retirement on a silver whistle which he had won for good shooting, and which he had used for giving signals instead of his nickel-plated whistle. He had noticed that the German officers used silver whistles which gave a note similar to that of his own. Further investigation showed that he had other delusions of a similar nature, one being that people thought him responsible for the railway accident near Edinburgh last May, because his troop train was due to go by that spot about the same time, and he had mentioned this to friends. He thought possibly a German spy had heard this and brought about the wrecking of the train, thinking it to be the troop train. Mental analysis revealed the memory that at the age of 12 the patient had been falsely accused of stealing pork-pies from a shop, and had been brought up before the magistrate for this offence. He succeeded in proving an alibi, but the incident made a deep impression on his mind and worried him for some years. Analysis of his dreams and of outstanding events

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in his life tended to show that this incident of the false accusation was the beginning of that tendency towards delusions of being falsely accused which culminated in the mental breakdown at Mons. After a two months' analysis the patient began himself to see the connexion between the mental effects of the various events of his past life, and the autognostic method produced a remarkable improvement in his mental condition. When I left him last month he was quite free from delusions, and showed a great improvement in his general mental grip.

Finally, I should like to draw attention to the curious fact that very few cases of mental breakdown seem to have occurred among the troops at Gallipoli at the time of the first landing, in spite of the awful slaughter. During the first two or three weeks after this landing I only saw about half a dozen cases of nervous breakdown in the Seventeenth General Hospital at Alexandria. One might attribute this partly to the protective influence of the sthenic emotions which actuated almost all the men at the time. The prolonged strain of trench fighting under high explosive shell fire is the chief cause of nerve shock, and this factor was absent at the beginning of the Gallipoli campaign.

DR. STANSFIELD.

The psychic effects which have been found accompanying shell shock are such as are ordinarily met with in the practice of psychiatry, and are then usually the outcome of stress producing exhaustion in a subject with poor resistance, owing to inherent or acquired defect.

The sustained mental tension necessarily associated with life in the firing line and its vicinity, kept up without intermission for two and sometimes three days at a time, with little or no sleep, is sufficient in itself to produce marked psychic disturbance in the most robust—varying with the individual—from dullness, retardation, and lethargy to severe depression with illusions and hallucinations, particularly of sight and hearing. In the case of the psychopathic it is therefore to be expected that the more profound lesions will be developed.

One is struck by the similarity of symptoms, in some cases labelled "shell shock," to those frequently found in dementia præcox—apathy, retardation, amnesia, and aphonia—so much so as to cause one to wonder if the primary and essential causes of this condition had not already been at work, and whether the stress in the trenches and under shell fire had only expedited the attack.

The most instructive and interesting cases to work out will be those in which there was no previous psychopathic tendency.

I have two cases of special interest to bring before you. One a case of amnesia following projected suicide, and one of protracted mutism:—

Case I.—P. M. M., aged 30, single, born in the West of Scotland, of a Scottish mother and an Irish father. Infancy and early life spent in very poor surroundings, and very suggestive of the scenes depicted in "Children of the Dead End." Enlisted at the age of 18½; served in South Africa and afterwards in India. Whilst in India he frequently became unduly tired after gymnastics, and he developed the idea that he was physically unfit, and on reading a book, "The Open Question," by Robbins, in which suicide is freely discussed, he decided that, without means, life for him was not worth living. He decided to complete his twelve years' service in the Army; then to have a real good time with the money he would have accumulated, and then to commit suicide. This idea he kept up for approximately five years. On completing his service he carried out his programme, taking about a month to get through the money he had accumulated. He then removed all evidence of identity from his clothing, &c.; left his bag with a man at the Union Jack Club; put a razor in his pocket, and walked out of London till he came to a common, intending to commit suicide. He was found on the common, taken in charge, and handed over to the parish authorities, being unable to speak or account for himself. The aphonia had passed off when he was brought to the hospital, but he had completely lost all memory of his past life. He was unable to give his name or any account of himself. He could perform all actions which were automatic, but otherwise was like a child. He could not name simple things such as pen, key, &c., but when told remembered their names. He had forgotten how to read and write, and even the letters of the alphabet were unknown to him. This condition obtained for seven weeks. He retired to bed one night in the condition described, and he awoke next morning to find his memory had returned. He was immediately able to give a very full detailed account of himself and his past life. He has, however, no recollection of the interval between lying down on the common outside London and finding himself in bed in the hospital here on the morning when his memory returned to him seven weeks later. In other words, there is a complete blank in his memory of seven weeks.

Case II.—The second case, W. G. W., aged 36, served in the Boer War and afterwards in India. Was invalided from India to Netley Hospital, and afterwards sent on to me. When admitted to Bexley he could not be induced to speak, but would answer simple questions by writing on paper, and would, by movements of his head, indicate "Yes" or "No." He did not appear to understand where he was, and would point and gesticulate when interviewed, smiling somewhat inanely. He remained in this condition for just over twelve months, when one day, when at the dinner table, he suddenly asked for a drink of water. He was then able to give a full account of himself. Before leaving for the Boer War he used to hear voices speaking to him, and saw all kinds of objects before his eyes, but could not be induced to tell the nature of these objects. He said that he would not go sick as he feared the other men would

think he funk'd the War. When at the Front he felt well for some considerable time; then he began to hear people saying things against him. When he asked them why they made certain statements they denied having done so. He complained of pains in his head at that time—things "pecking" at his head, which he thought was caused by men about him. When he left South Africa for India he thought people on the boat were chasing him to get his money. In Lucknow he heard himself accused of theft, and heard it said that he was going to be hanged. He told these things to the doctors, and as they did not believe him and made him out "in the wrong," he decided not to answer any questions in future but to suffer silently. He made slow but steady improvement, and was ultimately discharged recovered.

Both these men were very good musicians, and for some time were valuable members of the Institution band.

The matter-of-fact way in which the functions of speech and memory were resumed rather tends to exclude any suggestion of hysteria; and it is in keeping with numerous instances which I have observed after prolonged aphonia, where in one case the patient suddenly spoke when he accidentally dropped a big bundle of garments he had been struggling to hold, and in another instance the man, when practising in the cricket field at the net, shouted "Damn" when his middle stump was taken—the barrier in each instance, when broken down, not having been put up again.

The prognosis, even in the most severe cases, is, I believe, distinctly good, provided there is no neuropathic or psychopathic basis; but if the latter obtain, it varies with the extent of the lesion—in the severe cases being distinctly bad.

The treatment should be based on rational lines—absolute rest and quietness, with all the sleep possible, preferably with agreeable surroundings in the country; a nourishing, easily assimilated diet; appetite stimulated, if necessary, by simple tonics; little or no alcohol, and a watchful eye being kept on the excretory functions.

From my experience, I do not place faith in hypnosis or auto-suggestion, particularly in the early stages of treatment. Duration of treatment necessary varies from a few weeks to a few months.

I believe a great deal could be done towards reducing the number of these cases, and the severity of the attack, in those which do occur if commanders in the field could be induced to impress upon their men the importance and necessity of taking rest and sleep whenever possible; rest in the horizontal position being of great value by reducing the blood-pressure and thus relieving tension, even if absolute sleep is not obtainable. They should certainly make up their sleep account directly they are brought out of the firing line.

Note.—This Discussion was adjourned to the meeting of the Section of Neurology, held on January 27, 1916.

Sections of Psychiatry and Neurology.

Section of Neurology.

President—Dr. JAMES TAYLOR.

(January 27, 1916.)

ADJOURNED SPECIAL DISCUSSION ON SHELL SHOCK WITHOUT VISIBLE SIGNS OF INJURY.¹

Captain WILFRED HARRIS, M.D.

THERE are different varieties of shell shock. These affect naturally phlegmatic as well as congenitally neurotic individuals. It proves that prolonged mental strain and hard work may break down the nervous stability of even the hardest individual, especially if much under shell fire. Broken sleep, irritability to noise, forgetfulness of appointments and engagements, and inability to concentrate attention on reading or writing, are prominent features of most cases of shell shock, and the recovery from them is apt to be slow. In one case, after slow and continuous improvement for some months, the man became as bad as ever after experiencing a vivid dream of the battle he had been through. In another patient, who had previously been in perfect health, the characteristic symptoms of disseminated sclerosis followed immediately after the bursting of a shell some few feet above his head a month ago. Coarse nystagmus, intention tremors, syllabic articulation, incontinence of urine, and extensor-plantar reflexes were still present, though his mental condition, which was somewhat emotional, was said to have

¹ The first part of this Discussion is reported in the Proceedings of the Section of Psychiatry for January 25, 1916.

improved considerably. The possibility of numerous small lesions throughout the cerebrum as the result of the intense atmospheric disturbance following the explosion must be considered as a likely event. The patient had not been rendered unconscious by the explosion, and he is quite certain that he did not suffer in any way from being gassed as the result of it.¹

DR. COLLIER.

I consider that the conscious realization of the explosion and of its results, the severe sensory stimulation, and the remembrance of these, are the essential factors in the production of the train of functional symptoms under consideration, and that cerebral concussion stands in an antagonistic position to the psychical trauma. For if severe concussion produces immediate unconsciousness at the moment of the explosion, there can be no psychical trauma, and there is no remembrance of the event. In my experience, those cases in which an immediate loss of consciousness occurs suffer with the symptoms of cerebral concussion only and do not develop the functional symptoms. The investigation of this matter is difficult, since the patients always refer to the commonly occurring amnesia from psychical trauma as "unconsciousness."

I do not think that psychopathic and neuropathic antecedents are of importance as determinants of functional manifestations following shell shock. What seem more important are the proximity of the explosion and the violence of the sensory effect, provided consciousness be retained. Major Mott has referred to epilepsy as occurring only in those who have previously had fits, or in whom there is a family history of this disease. So far as epilepsy has resulted from shell shock, I am convinced that there are many cases in which no previous taint is present. This is probable from the important causal relation of fright to epilepsy, and it is important from the point of view of allotting compensation. "Fugues," or "wandering attacks with loss of memory," have occurred in some of my patients; there is a remarkable regularity of occurrence in the initial symptom of these attacks—a bursting feeling in the head, which increases until the loss of memory takes place.

The very low blood-pressure that obtains in cases of shell shock is remarkable. In only a very few cases has a high tension been noted, and such cases seem to be longer lasting and more resistant to treatment. Functional blindness has been very rare in my experience.

¹ He is now (February 20) practically quite recovered.

Complete functional deafness has occurred in one case. This patient had a similar attack some years ago when, as the result of an emotional shock, he became completely deaf for some months, and learnt to be a skilful lip-reader during his period of deafness, from which he recovered spontaneously. As the result of shell shock his symptoms have recurred. This patient converses so perfectly that it is difficult to believe that he is deaf. He cannot, however, be aroused by sound, and he can read perfectly the lips of anyone who speaks silently at a distance of 20 yards.

There have been a large number of cases of deafness, with mutism or lesser degrees of aphonia. These patients have been all alike with regard to the disappearance of the deafness prior to that of the aphonia, and as regards their ready facility in reading and exteriorizing written speech. One patient is worthy of a record as demonstrating the functional disregard of sound. He is a very intelligent man and was completely mute, and paid no attention to sounds. In the course of a written conversation he was asked if he heard anything when he was spoken to. In reply he wrote: "I can hear your voice quite well, Sir, but I cannot gather anything from it." This patient recovered spontaneously. He first began to recognize the last word of a sentence addressed to him and to repeat it audibly, and soon recovered his faculties of hearing and speech.

In the treatment of aphonia I have tried the effect of light etherization, which was suggested and used with success by Dr. Bastian many years ago. This has been successful in a few cases, but in the more severe cases it seems powerless to remove the resistance, and the patients go under the anæsthetic without any vocal excitation. The method, moreover, has the disadvantage that it may produce resentful emotions and loss of confidence on the patient's part if not successful. The most remarkable success in the treatment of the aphonic patients under my care has been brought about by another patient—an Irishman whom Nature has endowed with such a face and personality as make a perfect jester. He has also the faculty of moving his ears as a mule does. He would stand in front of the dumb patient and perform such comicalities of facial expression as in every case to force the patient into fits of laughter, with immediate restoration of speech.

I regard Major Mott's denunciation of massage and electrical treatment of the functional manifestations of shell shock as of too sweeping a nature, for, while the long continuance of such treatment in cases which do not soon show improvement is to be deprecated, there are undoubtedly cases in which these measures have greatly aided in recovery.

Mr. WALTER H. JESSOP.

I have had under my care numerous cases of temporary blindness following the explosion of shells; these cases were more often met with at the beginning of the War. The blindness, which was as a rule nearly complete—in some cases not even the flash of a match could be seen—lasted under a week, and generally about two or three days. Both eyes were always affected, the pupils were active to light, and the ophthalmoscopic appearances normal. The fields of vision were sometimes contracted, but more often normal; in only two cases did I find a spiral field. Two curious cases of fields of vision may be mentioned. The first showed complete bitemporal fields, which lasted for two days, when the fields became normal. The other was a case of complete homonymous hemianopsia, which persisted for some weeks, but turned out to be true and very clever malingering. Blepharospasm and photophobia were often present. In all my cases the recovery of vision was complete after some time; the treatment was by kindness, commiseration, and suggestion. The best results followed wearing dark glasses for a fixed and accurate time—say three days, six hours, and twenty-nine minutes. One case did not yield to the glass treatment, but responded on the night nurse waking him up suddenly at 2 a.m. with the remark, "Now you can see," by the answer, "Yes, as well as I ever did in my life." These shell shock cases can be fairly easily distinguished from the true malingeringer by both eyes always being affected and by their not being caught out by the usual vision tests for malingering. I have generally used the words "subconscious malingering" for the condition, feeling sure from tests, &c., that the patients are not conscious of their true condition. These cases may be explained by von Monakow's diaschisis theory. The windage of the shell, or in some cases the actual blows from falling down or violence, affecting the cortex cerebri, produces unconsciousness as the "initial" shock. As this passes off, loss of sight becomes the "regressive" phase, followed by complete recovery as the "residual" phase.

Dr. CAMPBELL THOMSON.

I am interested in Major Mott's observation that the return of memory for music frequently takes place before that of other memories, as this is in accordance with a case that has come under my own observation. In my case certain experiences of the man's life were lost—e.g., recognition of relatives, places, and animals; while others

(e.g., reading, writing, and music) remained. It thus seems that the memories which one would expect to be the more deeply impressed on the mind were lost in this instance; while those acquired later by educational processes were retained. Does Major Mott consider it likely that a functional type of cleavage of memory can be recognized, as opposed to a loss of memory from progressive organic disease, just as the anæsthesias and paralyses of the neuroses differ from those produced by anatomical lesions?

Dr. DUNDAS GRANT.

In view of the limitation of time I shall confine myself to a few remarks concerning mutism, stammering, and deafness resulting from shell shock and similar incidents of warfare.

In the treatment of *mutism* I deprecate the employment of violent measures during the period of exhaustion, as the use of strong faradaic currents has been known to be detrimental. When the exhaustion has passed off, I think their use quite justifiable. In the earlier stage I advocate the mildest measures. I look upon these patients as having, in a manner of speaking, forgotten how to speak. Being familiar with the method of teaching the subjects of deaf-mutism, my opinion is that it may be applicable to these cases. The first step is to place the back of the patient's hand under the teacher's larynx, so that he may feel the vibrations produced by the utterance of voice; the hand is then transferred to the patient's own larynx and he is encouraged to produce the same feelings, and ultimately the same sort of sound, in the larynx. From this he is gradually led to alter the shape of the mouth cavities for the production of vowels and, later, of consonants. In some cases of concussion-mutism the voice is restored at one sitting, but in others it is a gradual process. There is no simulation about the inability to speak; the patient's anxiety to do so is unmistakable, and his delight at the gradual acquisition of words and phrases quite confirmatory. In two cases I have observed that the voice returns before the hearing, and I think that the act of uttering sound has helped to arouse the hearing faculty. In one very well-marked, long-standing case at the West End Hospital for Nervous Diseases, I put these measures into practice, and fortunately with such excellent result that the house surgeon has adopted the method with great enthusiasm, and by its means has restored the function in five or six cases.

With regard to *stammering*, which seems to be due to a complex

of inhibitions and exaggerated effort, the anxiety of the patient to overcome it is unmistakable, but the more he strives the less he is able to succeed. The efforts are often accompanied by the utmost fatigue. It is therefore necessary in these cases to proceed gently and gradually. It is well known that the so-called curers of stammering teach patients to practise little tricks such as twirling a button, or snapping the fingers, or making some muscular movement before beginning to speak. These are generally totally unconnected with phonation, but there is one muscular action which serves the same purpose of diverting some of the patient's anxiety, and which is, in point of fact, part of the mechanism of speech—namely, the expansion of the lower part of the chest; and in several cases recovery has been greatly hastened by the practice of this little manœuvre before speaking. The Behnke method consists to a large extent in the practice of breathing exercises, and I attribute a great deal of its success to the fact that its practice diverts the patient's attention from his impediment.

In regard to *psychical* deafness there are some points in the diagnosis to which I shall refer. Of its genuineness there is, as a rule, no question, and this is proved to my mind by the fact that the patients sometimes acquire lip-reading with considerable rapidity. I am of opinion that the acquisition of lip-reading is unquestionable evidence of a high degree of deafness. The diagnosis from simulation can generally be made by means of various checks, well known to aural surgeons and "compensation" experts. As compared with deafness due to labyrinthine causes, the psychical deafness is to all appearances more absolute. Again, in labyrinthine deafness, hearing is especially bad for the highest pitched tones. Further, there is often a simultaneous involvement of the vestibular labyrinth, as indicated by loss, or diminution, of reaction to Bárány's test, though frequently the acoustic labyrinth suffers without the vestibular apparatus being involved. In several instances I have tried another reflex which I think will prove of value, although more experience will be necessary to decide this. It is the dilatation of the pupil which follows the sudden blowing of a whistle without the patient being aware of it; in several cases of labyrinthine deafness this was absent, but in others which seemed to be of psychical origin the reflex was present, although the deafness was absolute or nearly so. In the dullness of hearing due to exhaustion, which shows the tuning-fork indications of nerve-deafness, there is usually preservation of hearing for the highest pitched tones. Labyrinthine and psychical deafness may be combined, but the labyrinthine factor is generally unilateral.

Dr. FEARNSIDES.

During recent times shell shock without visible injury has been a diagnosis used loosely, and frequently applied to label any functional nervous affection manifesting itself in a patient who has been subject over a period of weeks or months to the strains and stresses of active foreign service. In order, therefore, to collect together some definite figures bearing upon the ætiology of the cases so described, I have analysed a series of seventy consecutive admissions to the Hospital for Epilepsy, Paralysis and other Diseases of the Nervous System, Maida Vale, of patients suffering from "functional nervous diseases" arriving directly from France with "special" tickets.

In twenty-three of these seventy cases the actual shell shock has been preceded by a period of nervous instability with a complaint of general weakness, tremulousness, inability to sleep, night starts or other symptoms of mild nervous derangements, and then acute collapse following upon the explosion of a shell in the near vicinity, burial in a dug-out, or some one or other of the various modes of onset detailed by Major Mott. Of these twenty-three cases seven, or 10 per cent. of the whole number of admissions, had previously suffered from "traumatic neurasthenia," which in six cases had been the subject of litigation, and four others gave either a history of persistent "nervous debility" in youth or of a previous "breakdown." In four of these patients where no history of a previous "breakdown" was obtained a bad neuropathic or psychopathic family history was detailed. The average age at the time of admission to hospital of those giving a history of previous traumatic neurosis was just over 31, the youngest being 25; whilst of those giving a bad personal or familial history the average age was under 26, the youngest being 19. Only in eight of these twenty-three cases was no history of previous neuropathic tendencies obtained in answer to queries, and it is noteworthy that of these eight cases, five occurred in non-commissioned officers who had been at the Front on the average for more than twelve months, and had apparently broken down through lack of adequate rest; the ages of these eight cases averaged 34.

Four patients, examples of shell shock, detailed how their general health went wrong after an "influenzal" or "diarrhœic" attack, and then a shell burst near them and they broke down completely, whilst one had suffered for ten days from a recurring toothache before he "became nervous during a bombardment." In three of these five cases

the dilatation and irregularity of the heart, accompanied by subjective symptoms of breathlessness and palpitation, were noteworthy, and a study of these cases brought forcibly to my mind the close relationships which exist between shell shock and soldier's heart.

In six cases many of the manifestations could be related to an infection with the *Spirochæta pallida*, for in them the Wassermann reaction was found to be positive at least in the serum, and in several also in the cerebrospinal fluid. The complaints and histories of the patients in these cases differed in no marked way from those obtained in straightforward examples of true shell shock, and I would like to suggest that in all cases of shell shock the Wassermann reaction in the serum should be tested; for in my experience cases of shell shock giving a positive Wassermann reaction which have held fire when treated symptomatically often improve in a most extraordinarily rapid manner when treated with antisyphilitic remedies. In this respect cases of shell shock differ in no gross way from cases of "syphilitic neurasthenia" seen in civil practice. In addition to these six cases two examples of dementia paralytica and one of tabes dorsalis were admitted to hospital with "green tickets," having developed signs and symptoms whilst on active service. Thus in nine cases out of seventy admissions syphilitic disease of the nervous system accounted for some at least of the manifestations alleged to be consequent upon shell shock.

Three patients exhibiting manifestations which closely resembled those of shell shock had suffered from gunshot wounds of the trunk or extremities, and I have seen several examples of patients suffering from gunshot wounds of the head, in whom the dura mater had suffered no grave visible injury, complaining of so-called functional nervous disturbances.

In twenty-five cases symptoms had come on suddenly after, as far as could be ascertained, a period of good health; of these twelve showed gross disturbances of motor functions—hysterical postures, paralyses, and the like, usually accompanied by hysterical anæsthesia—and thirteen exhibited more purely psychical manifestations; in one a rupture of the drum of the right ear at the time of the explosion complicated the psychical phenomena. The average age of the patients in these twenty-five cases was 24.

A point of psychological interest, I think, is the relation of the causative accident to the immediate previous history of the patient. Most of the cases in whom symptoms have come on acutely occurred at a time when the men were going to trenches from their billets, or when

shells were arriving spasmodically and occasionally rather than in rapid succession. When a man's "wind is up" he is more receptive of a psychical trauma. Dr. Mott spoke of the relations of gas poisoning to the development of shell shock, but the cases which I have had under care did not seem to me to bear out any intimate relationship between gas poisoning in gas attacks and shell shock, nor could I get any definite clinical evidence to suggest that the gas from the poison-gas shells was a serious factor. Speaking of gas attacks, when the men in April, May and June, 1915, were expecting such an attack, regimental medical officers with whom I have talked have told me that a chance shot at these times more frequently broke the men down than did such shots under ordinary circumstances.

Dr. LEONARD GUTHRIE.

There are various modes of treatment of functional neuroses, and of hysteria in particular. I believe that all forms of treatment involve the employment of either suggestion and persuasion or of some novel and unexpected emotional or physical shock. The latter method is known as "taking the patient by surprise," and is chiefly successful in cases of hysteria.

Major MOTT (in reply).

I am very interested in the remarks of Major McDougall and Captain Brown on the value of hypnosis in the treatment of cases of functional neurosis and psychosis. I am of opinion that a careful selection of cases is necessary, and I should only be inclined to try it on cases that did not yield to suggestion and other modes of treatment, the most important being diversion of the mind by games, amusements, and occupations in the open air. I agree with Major McDougall that cases of shell shock without visible injury may be associated with severe retrograde amnesia. But unless there was definite evidence of no other factor than the physical force generated by the explosion, I would rather attribute it to the patient having been "sandbagged" or, while buried beneath the blown-down parapet or roof of a dug-out for some time, having been exposed to enforced inhalation of noxious gases, notably CO.

I am very interested in the case of Captain Wilfred Harris, in which there were signs of disseminated sclerosis. A brain was sent to me taken from a man who had been buried by shell-fire, and throughout

there were multiple punctate hæmorrhages in the white matter. Had he lived there would have been islands of sclerosis.

I am glad to hear Dr. Head's remarks emphasizing the importance of fear in the production of what is termed "shell shock," and I agree with him that the term is not correct; for it covers a number of factors in the production of functional neuroses and psychoses observed in soldiers. I am convinced that an individual with a timorous disposition is more likely to suffer from a rapid breakdown than others, and I cite the following case, which has been under my care, as showing the importance of emotional shock in the production of unconsciousness and a subsequent nervous breakdown:—

A corporal in a Highland regiment was sent out with a company to repair the barbed wire in front of their trench. While engaged in this operation a big shell burst, blowing him some distance into a hole. He scrambled out and then saw his comrades lying dead and wounded around; he lost consciousness, and did not know any more till a fortnight later he found himself in Boulogne. He was admitted to the Fourth London General Hospital; he presented the appearance of abject terror, and moved his arms in a way suggesting that he still saw the dead lying all round. He muttered continually: "No send back," "Dead all round." The loss of consciousness did not occur till he saw the terrifying sight. Of this he dreamed continuously, and only when the dreams passed away and normal sleep returned did this patient lose the expression of terror.

In reply to Dr. Collier—who has evidently misunderstood me when he asserts that I said "*only*," whereas I said "nearly always" in epilepsy a previous condition or predisposition existed—careful inquiry in my cases regarding family history and personal history leads me to believe that in the great majority of cases of epilepsy there has been a history of epilepsy, *petit mal*, or neuropathic predisposition, and that the shell shock aggravates a pre-existing condition. In support of this statement I find that, out of twenty-five cases of epilepsy, in twenty there was evidence of either a former epileptic condition or of a predisposition; of the remaining five there was a history of head injury in two prior to joining the Army. I am indebted to Dr. Cicely Peaké's very careful notes for this information. I do not wish for one moment to imply that a predisposition to neurosis is necessarily present in the individual prior to active service in these cases of shell shock. Indeed, I have remarked that cases occur in men who are neuro-potentially sound, and these are usually men who have gone through months of active service at the Front before the breakdown occurs. Even the

strongest nervous system cannot withstand a prolonged cumulative effect of shell-fire and trench warfare without nervous exhaustion.

Again, Dr. Collier stated that my denunciation of massage and electricity was of too sweeping a nature, "for while the long continuance of such treatment in cases which do not soon show improvement is to be deprecated, there are undoubtedly cases in which these measures have greatly aided in recovery." But what did I say? These are the words, which are not a sweeping denunciation: "Prolonged massage and electricity of all forms are better avoided, *as a rule*, unless as a means of suggestion of cure by their use," and of the truth of this I am absolutely certain.

Dr. Collier remarked I referred to defects of vision and blindness along with other sensory troubles as if that were a common condition. In my opinion it is a rare condition of functional neurosis. I did not knowingly imply that I had seen many cases; there have been two or three. Naturally such cases would come under the notice of ophthalmic specialists, and I am much interested in hearing Mr. Jessop's remarks, which indicate that these conditions are not rare. However, comparatively to deafness and hyperacusis they are undoubtedly uncommon.

In reply to Dr. Campbell Thomson, I am much interested in hearing he has had a case in which the musical memory had returned before other memories, and his explanation of cleavage of memory is worthy of careful thought and consideration.

In reply to several speakers, regarding the value of suggestion and hypnosis, I am of opinion that strong suggestion, by giving the assurance that there is no organic disease, is very necessary. A careful physical examination of the patient, followed by an emphatic statement that he will get quite well, gives assurance, and will often serve to uproot a "fixed idea." The difficulty comes, however, with patients who have a "fixed idea" that they do not want to get well. A patient who was said to be suffering with paraplegia, and who was treated for months by massage and electricity, was immediately cured by strong suggestion, also another who had had violent tremors for months. In another case the patient was told by the panel doctor that he was paralysed in his left arm, and for weeks he had refused to move it. But on being informed that electrical examination had proved there was no paralysis and if he did not move it the next visit he would be court-martialled, he was seen buttoning up his trousers with it five minutes later.

Dr. Fearnside remarked that he had not obtained evidence showing that "gas" had any effect in producing symptoms, but I was not

xliv Mott: *Shell Shock without Visible Signs of Injury*

referring to the irritating effects of noxious gases—e.g., Cl.—but to the effects of carbon monoxide caused by imperfect detonation of high explosives in closed spaces such as trenches and dug-outs, in which soldiers may lie for some time buried, and which, being inodorous, would not be recognized.

Section of Psychiatry.

President—F. W. MOTT, Major, R.A.M.C.(T.), F.R.S.

(March 28, 1916.)

The Psychology of Rumour.

By BERNARD HART, M.D.

THE subject of "rumour," though at all times a fascinating field for the psychologist, possesses to-day an exceptional importance and interest. Recent history has furnished an overwhelming demonstration of the fallibility of human evidence, and has provided a mass of material which should yield a rich harvest to the scientific investigator. The aim of the present paper is to indicate the results which in the past psychology has already achieved in this field, and the bearing of those results upon the problems of the present day.

Rumour is a complex phenomenon consisting "essentially in the transmission of a report through a succession of individuals. It may be provisionally regarded as the product of a series of witnesses, each of whom bears testimony to a statement imparted to him by his predecessor in the series.¹ The reliability of a rumour depends, therefore, upon the accuracy with which each such statement is transmitted, and ultimately upon the accuracy of the report furnished by the first member of the series, who is assumed actually to have seen or heard the event in question. This latter factor, the testimony of the actual witness of an event, is what the law terms "evidence," and it is clear that an examination of its psychology must precede any attempt to attack the more complicated problem of rumour.

¹ It is necessary to emphasize the provisional character of this definition. We shall subsequently find that it is incomplete, and that it requires considerable modification (*vide* p. 13).

The first scientific investigation of evidence seems to have been carried out, not by psychologists nor by jurists, but by historians. The methods of historians in estimating the value of evidence have undergone a considerable change in modern times. Formerly they accepted the moral character of a writer as the test of his reliability; if his character was known to be good then the statements which he made were held to be accurate. They debated whether their witness was honest or dishonest, whether he spoke truth or was deliberately trying to mislead for certain definite conscious ends. If this question was decided in the witness's favour then all his evidence was accepted. Now, however, the reliability of each individual statement is separately estimated. The moral character of the witness remains, of course, an important factor, but the historians also take into account every possible condition which may have exerted an influence upon the particular statement under examination: the source of the witness's knowledge, the time-interval separating him from the events in question, his views and prejudices, his profession, religion, and political party. They consider, moreover, not only whether the witness is deliberately lying, but whether and how far he is unconsciously perverting the truth owing to the action of the factors just mentioned. In recent years attempts have been made to codify the rules to be observed by historians in the estimation of evidence, and a considerable literature on this subject now exists, amongst which may be particularly mentioned the works of Ernst Bernheim [2].

Among professional psychologists the first definite step in the direction of investigating the psychology of evidence seems to have been taken by Binet [3], who in "*La Suggestibilité*" (1900) called attention to "the advantage that would accrue from the creation of a practical science of testimony." The suggestion was taken up enthusiastically by Stern, of Breslau. Stern [15] founded a school of experimental psychology whose energies were almost entirely devoted to the psychology of evidence. Their work was in the main experimental, and they carried out a prodigious amount of painstaking research. One can make no attempt to give any adequate account of this work here, but it will be of interest to describe the general methods employed, and to give a short résumé of the principal conclusions reached.

The methods adopted all consist essentially in submitting a predetermined experience to a number of subjects, subsequently obtaining from the latter a report of what they have experienced, comparing the reports with the original experience, and finally collating and evaluating

the results thereby achieved. For example, a picture shown is for a defined period of time, and after a fixed interval each observer is required to give evidence as to the nature and details of the picture he has seen. The time-interval between observation and report may be varied from nothing to several weeks. The report is obtained by two different methods—narrative and interrogatory. In the former the subject is asked to write out as fully as possible all he has seen. In the interrogatory method a number of questions are asked by the experimenter, designed to cover in their entirety all the details of the original experience. The subject is asked, for example, "Were there any animals in the picture?" "What colour were they?" The questions are carefully constructed and classified into simple inquiries devoid of any suggestive implication—e.g., "What is the size of the picture?"—and "leading questions" containing suggestive implications of various degrees—e.g., "Has the man a brown coat?" The interrogatory method corresponds essentially with the "cross-examination" of our law courts. Each detail of a report obtained by these methods is graded according to the subject's assurance of its reliability. The degrees of assurance generally distinguished are "complete uncertainty," "hesitancy," "certainty," and "attestation." "Attestation" means that the subject is prepared to swear to the truth of the evidence in question.

The most important general result of the experimental investigations conducted along these lines is that they upset in the most definite and complete manner two naïve views widely held by the laity: (1) that evidence given with the best knowledge and honesty is a correct reproduction of actuality; (2) that evidence which is shown to be false must be due to deliberate lying, or at least to culpable carelessness.

Experiment shows us that completely correct reports are not the rule but the exception, even when the report is made by a competent observer under favourable conditions. It must be clearly understood, moreover, that this statement remains true if only those details are taken into consideration of whose accuracy the reporter is certain. Borst [5] found in 240 reports only 2 per cent. of errorless narratives and 0.5 per cent. of errorless depositions—i.e., reports obtained by the interrogatory method. The average reporter, when no suggestive questions are employed, exhibits a coefficient of accuracy of roughly 75 per cent. In other words, only 75 per cent. of the items of which the reporter is *certain* are in fact accurate. Moreover, attestation does

not guarantee accuracy. Although the number of errors in sworn testimony is considerably less than that in unsworn testimony, they may nevertheless amount in the former to as much as 10 per cent.

A detailed examination of the results obtained by these investigations yields a large number of interesting facts, of which the following may be selected for special mention: The effect of increasing the time-interval between the observation and the moment when the witness is called upon to give evidence regarding it is that, though range and accuracy are both diminished, assurance is not equally affected, but shows a surprising constancy. This statement, rendered in non-technical language, means that, though the number of details remembered and their accuracy are both diminished by lapse of time, the witness's belief in the truth of his evidence is but little affected. From this it may be concluded that assurance and readiness to swear to the truth of the evidence given depend upon the "personal equation" of the witness rather than upon freshness of memory. A second interesting fact established by the experiments is that, if the interrogating method is employed instead of the narrative method, the range is increased but the accuracy is greatly diminished, that is to say, more details are remembered but fewer are truthfully reported. This is, of course, due to the suggestive influence exercised by the questions asked. The diminution of accuracy when definitely suggestive or leading questions are used is evident enough in adults, but in children this effect is very marked indeed. For this reason evidence given by children should only be accepted with the greatest caution; their range is small, their accuracy smaller still, while their assurance is relatively very high.

It will be seen at once that these experimental results are of very great practical value, and that they establish conclusively certain basic facts which are of fundamental importance to the psychology of evidence. Stern and his school, however, do not get much beyond this. Their work, except here and there, presents us with no dynamic view of the forces responsible for the facts they describe, no conceptions which enable us to understand *why* these things do and *must* take place.

We get some illumination in these respects, however, from a third group of investigators, who approach the subject from a different point of view and with a very practical aim—the jurists. The lawyer must obviously have a considerable interest in the psychology of evidence, and legal authorities have from time to time during the past two centuries shown that they possessed at any rate some inkling of the

principles ultimately laid down by Stern and the Breslau school. Jeremy Bentham concerned himself with the subject in considerable detail, and later legal writers have dealt with supposed perjuries and the danger of suggestive questions. The first comprehensive and detailed work from the legal side on the psychology of evidence, however, would seem to be that of Hans Gross, the well-known criminologist.

Gross [9] points out that the psychology of evidence involves not only a memory-process and the question of the fidelity of that process, but also the processes of perception and registration which take place at the moment of the occurrence reported upon, and he holds that even more weight is to be ascribed to these latter processes than to the former. He lays stress on the personal equation of the observer, and shows that the same objective event may be very differently perceived by different observers.¹ What a man sees depends not only on what is actually presented to him at the moment, but on perceptual additions due to prior knowledge and interests. He tends to group an event egocentrically, to overweight the factors which arouse his interest and to neglect others. This explains the paradox that an observer who knows nothing whatever of a subject may be a better witness in a matter connected with that subject than one who is an expert therein.

Another legal authority, Heilberg [10], brings forward some further considerations of great value. He points out the important influence exerted by events intervening between the observation and the report, and shows how the accuracy of a memory picture which is constantly brought up and discussed may be perverted, owing to the action of auto-suggestion, external suggestion, and other factors, at least as much as a picture left in pure passivity. He explains by this principle the epidemic of false witnesses which so often occurs in the later stages of sensational trials. Heilberg, moreover, illuminates the path along which we must tread in our search for the psychological laws responsible for the perversion of evidence, in that he calls our attention to the influence exerted upon the witness by the solemnity of the court, the feeling that he occupies the centre of the stage and that his words are big with fate, and the consequent appeal to his vanity.

¹ Gross points out that we do not actually see what happens in a moment of time, but a combination picture, grouped from successive moments, and the mode of grouping may be different in different observers. Thus, if an event objectively consists of *a, b, c, . . .*, then one observer may perceive *abc, def, ghi*, another *bcd, efg, hij*, while a third may miss points and perceive *acd, fhi, kmo*. This conception seems to correspond with the "noetic form" of modern psychologists.

Some extremely interesting and stimulating observations are to be found in an article written by Stern in collaboration with his wife, entitled "Memory and Testimony in Early Childhood" [15], and consisting essentially of a study of the gradually developing mind of their own child. The points which we desire specially to mention are contained in a chapter on "False Witness in Children." Here the authors develop the important proposition that between lies and genuine perversions of memory there exists an intermediate group of phenomena to which they give the name of "pseudo-lies." The commonest variety of these pseudo-lies is the relation of some fiction which the child represents as an event which actually occurred. On one occasion, for example, a child informed her parents, after a visit to the Zoo, that she had stroked the bears, and became tearfully and stormily insistent when the veracity of her account was called into question. This, of course, is the phenomenon which we term "phantasy," and which is familiar to us in the day-dreaming of the adolescent. In the child, however, phantasy is not sharply distinguished from reality, and it tends to play with fictitious accounts of past events just as it plays with fictitious representations of the present. "While reality and make-believe in the life of the child are not yet distinct from one another, so also are truth and falsehood not yet distinct." A similar inability to distinguish truth from phantasy has been noted by Cramer [7] in the case of imbeciles.

So far as I have been able to discover, Stern does not seem to have applied these valuable observations upon the forces at work in the child to the problem of the psychology of testimony in the adult, nor to have realized that the adult is moved by precisely the same forces as the child, though of course less blatantly, and with their action modified by other factors.

The part played by phantasy in the psychology of testimony has also been dealt with by the historian, Ernst Bernheim [2]. This author has seen, moreover, the close relationship between the mechanism at work here and that underlying the evolution of myths and sagas, a problem to which we shall subsequently return. Bernheim also remarks on the influence exerted on the witness by the impulse to assign satisfying motives and to round off the story. When we pass from the question of testimony to that of the transmission of a report through a number of witnesses—i.e., from the question of evidence to that of rumour—the recognition of this factor becomes of essential importance.

Now if we review the various facts elucidated by the investigators we have so far considered, and endeavour to group them into a coherent

whole, we shall find that our task will be greatly facilitated by applying to those facts certain conceptions employed in modern psychology, in particular the conception of the "complex." We shall thereby be enabled, moreover, to obtain a considerable insight into the mechanisms responsible for the phenomena whose existence has been demonstrated.

A "complex" may be defined as a system of related ideas possessing a certain affective load which tends to produce in consciousness trains of thought leading in a definite direction. Thus we explain the circumstance that two politicians will arrive at diametrically opposite conclusions when presented with the same body of facts by saying that the train of thought is directed in the one case by a Conservative complex, in the other by a Liberal complex. It is held, moreover, that a complex may exert its action without the individual himself being in the least aware that his thinking is so directed. The individual may, indeed, believe the causes responsible for the conclusion he has reached to be quite other than they actually are. For example, each of the two politicians may honestly believe that his opinion is the purely logical result of a dispassionate consideration of the facts presented to him. The process of self-deception by which this erroneous belief is given a superficial plausibility is termed "rationalization."

This conception has proved extremely fruitful in both normal and abnormal psychology, and we can derive considerable aid from it in our investigation of the mental processes involved in testimony. It will be convenient to divide these processes into three stages—perception, conservation, and reproduction—and to examine in each stage the effects that may be produced by the action of complexes.

Firstly with regard to *perception*. It is an elementary commonplace of psychology that in every perception an endogenous factor is involved. When we perceive an orange, our percept consists, not only of the extensive yellow sensation which is all that is actually presented to us, but of an indefinite number of other factors, traces of former muscular and tactual sensations for example, which are added thereto from the store of our past experience. An endogenous factor of another kind is also present, however, whose activity accounts for the fact that perception is a selective process, and not a merely passive submission to sensations. We tend to pick out from the material presented by our senses the elements which are for some reason interesting to us, and to perceive only that in which we are interested. We may express this process by saying that our complexes exercise a selective action upon

our perceptions. Complexes may exert, moreover, an action which is not only selective but perversive, and our perceptions may not correspond with the objects actually presented to us. Thus we tend to perceive what we expect to perceive, to mistake the stranger entering our gate for the friend whose arrival we are awaiting, to hear the motor-car for which we are anxiously listening. We can explain in this way some of the evidence which was showered upon us at the time of the Russian rumour. I conversed personally with a soldier who assured me that he had himself seen trains filled with Russian troops passing along the line where he was on guard, and he described to me the tall bearded men and the unusual uniforms. The effects of complexes are proportional to their emotional strength. Hence there is a very grave danger of perverted perceptions in times of great emotional stress, a danger which must be carefully taken into account if, for example, we are called upon to weigh the evidence of even eye-witnesses of scenes occurring during the storm of battle or invasion.

Passing on now to the second process involved in evidence, *conservation*, we find further mechanisms by which perversion may be produced. Our memory-traces of events we have witnessed are subject to simple forgetting, whereby elements may be lost whose omission materially alters the picture, and to active forgetting, the process termed repression, whereby elements may be dislocated from their normal position or essentially altered in their character. The manner in which the unhappy experiences drop out of our memories of former holidays, and the illusory rosy light which so often shines upon the past, may be cited as examples of this latter mechanism. In it, of course, complexes play an important part.

In the third and final process, that of *reproduction*, there are yet other mechanisms tending to the perversion of evidence. Some are due to the suggestive power exerted by the personality of the examining counsel, and the form in which he casts his questions. Here belong, also, those effects produced on a witness by the particular circumstances attending the giving of his evidence, which have been insisted upon by legal investigators. Among such circumstances may be mentioned, for example, the solemnity of the trial with its paraphernalia, and the "centre-of-stage" feeling of the witness. The most prominent complexes here in action are the grandiose or "self-assertion" group. Hence arises an impulse which drives the witness to say something effective, to round off the story, and to fill in gaps—in fact, an impulse to stage his evidence so as to satisfy the canons of dramatic art.

Closely associated with these latter factors is phantasy, which may be regarded as affecting both conservation and reproduction. Phantasy is produced when complexes, instead of trying to achieve their ends by influencing the world of reality, obtain a partial satisfaction by the construction in the mind of trains of imagery, in which the ends of the complexes are, in imagination, abundantly realized. A simple example is the well-known day-dreaming of adolescents. Stern, in the paper on the child to which we have already referred, fully recognizes the important part played by phantasy in the perverted testimony given by children, but there is, of course, no doubt that this factor is fundamentally important in the adult also, although its action is not so obvious and unrestrained as in the child. Ogden [12] has pointed out that the essential difference between phantasy and memory does not appear to lie in any definite peculiarity of content, for intrinsically they are not clearly distinguishable, but that it is largely the problem before us with its directive tendencies upon which the practical distinction rests. Hence it is easy to understand that complexes, which occupy so important a position amongst the directive forces of the mind, may introduce phantastic elements into a memory-content without the interpolation being detected by the individual himself. As examples of the effect of phantasy upon evidence taken from the sphere of law may be mentioned false confessions and the often-noted appearance of false witnesses in the later stages of sensational trials. In everyday life examples are easy to find. Our alleged memories of the events of early childhood are often destitute of any but the smallest foundation in fact, and that lack of veracity in the recital of past exploits, in which a possibly undeserved pre-eminence has been attributed to the fisherman, is by no means to be regarded entirely as the result of deliberate lying.

Our understanding of the part played by complexes in the perversion of testimony will be deepened if we take into consideration the facts provided by pathology. We should expect to find here, of course, perversions which are more obvious and accentuated. This very accentuation, however, will enable us to grasp their essential character, and thereby to appreciate the presence, though in a far slighter degree, of precisely the same mechanisms in the normal.

For this purpose it will be convenient again to divide the psychological processes involved in evidence into the three stages of perception, conservation, and reproduction, and to examine the pathological variations occurring in each.

The pathological perversions of perception include hallucinations, illusions, and certain delusions of reference. All these are now generally regarded as being due to the distortion of perception by an endogenous factor, and this endogenous factor can be traced, in some instances at least, to the action of complexes, whereby ideas dissociated for some reason from the main stream of consciousness are, as it were, projected outwards.

It is a little difficult to distinguish sharply between the pathological variations of conservation and reproduction, and they may therefore be considered together. Here belongs the well-known phenomenon of paramnesia, of which examples frequently occur in many varieties of mental disease. Such, for instance, are the alterations in the memories of the past produced by a system of delusions, and the confabulations of the alcoholic or general paralytic. Most interesting for our present purpose is, however, the condition known as pseudologia phantastica, characterized by the relation of fictitious reminiscences. The patient, with an air of entire verisimilitude, will give a detailed and often elaborate history of his past life and experiences, which investigation shows to be wholly imaginary. Dr. Stoddart, in a paper devoted to the description of an extremely interesting case of this disorder, ascribes the symptoms to the presence of a morbid instinct for lying. I must confess that I do not find this theory very satisfactory. Such an instinct would presumably affect all the patient's statements, whereas in fact only certain statements are perverted, and the perversion is always in a definite direction. Dr. Stoddart's patient, for example, whom I subsequently saw at Long Grove, did not lie as to whether she had had beef or mutton for dinner, but only in the construction of a fictitious past which presented her as a distinguished, influential, and exceptionally interesting person. In this case the confabulations were evidently elaborate phantasies, whose creation we must ascribe to the activity of grandiose complexes, and it seems probable that a similar mechanism always underlies the manifestations of the disorder. Such a conception, moreover, enables us at once to understand the obviously close similarity existing between pseudologia and the ordinary day-dreaming of adolescents. This latter analogy has been noted by Risch [14] in the course of a very valuable paper wherein five cases of pseudologia phantastica are described. He finds in all cases certain common factors, among which may be specially mentioned an irresistible impulse to confabulate, with the consequent production of a peculiar feeling of pleasure, and a characteristic egocentric orientation of the

patient's trains of thought, so that the patient is himself always the hero of his confabulations.¹ The patients, in their pseudo-reminiscences, keep fairly well within the possible, and only the total bears the stamp of fabrication. Unlike the ordinary liar, they are untroubled by exposure, and merely stimulated thereby to further confabulations, or to some often ludicrously inadequate evasion. One patient, for example, when his story of exciting adventures in the company of a Russian count was demonstrated to be entirely fictitious, merely remarked, "But I have nevertheless often met Russians." So far as I can make out, Risch seems to think that the patient has a genuine belief in his own fabrications, although he endeavours to distinguish the condition sharply from dementia paranoides. It is, however, very questionable whether such a belief really exists. The reaction of the patient, if exposed, is quite different to that accompanying the demonstration of falsity of a delusion; the fabrication *can* be exposed, the delusion cannot. It would seem that in the pseudologia patient the condition, as regards degree of belief, is a half-way stage between the entire absence of belief found in the case of ordinary day-dreaming, and the full and absolute belief accompanying delusions. This half-way stage is difficult to define, but it is probably closely similar to the pseudo-lying observed in children, which Stern has so admirably described in the paper we have already mentioned.

Nearly allied to the phenomena of pseudologia phantastica are the well-known phantasies of hysterics. An hysterical patient under my care at University College Hospital presented a member of the staff with some obviously freshly cut chrysanthemums which, she said, had been sent to her by a relation in California. When doubt was cast upon her story, she endeavoured to substantiate it by the production of a forged letter purporting to come from the relation in question, in which reference was made to his gift. As a further example of hysterical phantasy may be mentioned the well-known false accusations of rape. The complexes underlying these cases are, of course, obvious.

The importance of the facts of pathology in considering the question

¹ Risch points out that these fabrications are related not only to the phantasies of the day-dreamer, but also to the trains of imagery which occur in the novelist and poet. He considers, however, that the patient plays the rôle of actor as well as that of author, whereas the novelist and poet play only the latter. This is, I think, a misapprehension of the psychology of the novelist, who, in many cases at least, obviously lives in the characters which he creates. As an interesting instance we may mention Mr. Arnold Bennett's "Clayhanger" and "Hilda Lessways," two novels in which the same scenes are described, in the first from the standpoint of the hero, in the second from the standpoint of the heroine.

of the psychology of evidence is clearly very great, for they serve to show to what an extent it is possible for testimony to be perverted by phantasy. At the present time numerous examples of these more extreme perversions are occurring, and are playing their part in the propagation of rumours. We may cite the case of the Scotch nurse, which attained considerable newspaper notoriety in the early days of the War. A young girl produced letters purporting to come from a hospital in France, describing the death of her sister after fiendish atrocities had been perpetrated upon her. The story was made public and aroused widespread indignation and sympathy, promptly cut short, however, by the alleged victim herself, who announced that she was alive and well, and had never left the confines of Yorkshire. Investigation showed that the girl had written the letters to herself, and that they were obviously the productions of hysterical phantasy.

It must be remembered, however, that these pathological cases are only extreme instances of the activity of a psychological process which belongs to the fundamental structure of the human mind, that the steps from the pathological to the everyday are easily graded, and that the phantasy which is responsible for the bizarre phenomena we have latterly been considering is made of the same essential stuff as the normal phantasies of the child and the quasi-normal phantasies of the day-dreamer. Hence it is easy to understand that this process in its minor grades may play a part in the perversion of evidence given by average men, and that it constitutes a factor which must always be carefully estimated when that evidence has to be weighed. Recent history has shown, indeed, that such perversions of evidence in normal men may, under suitable conditions, attain a growth hardly less luxuriant and phantastic than that we have described in cases which are definitely pathological. Except for recent history, we should, I venture to think, have unhesitatingly said that in normal people perversions could not occur to such an extent, and we should have drawn the line between normal and pathological far more definitely than experience has shown to be actually allowable.

So far we have dealt only with the question of testimony, that is to say, the report given by a witness on some event which he has himself seen or heard, or which he has himself experienced, and with the perversions to which his evidence is liable. Before passing on to the larger and more complicated, but closely allied, problem of rumour, we may pause for a moment and summarize the position we have been able to reach.

We have found, by the test of experiment, that a witness's report of his experience, even when given with the utmost honesty and conscientiousness, is rarely a completely correct reproduction of actuality, and that a proportion of the details, including even those of whose truth the witness is absolutely certain, are erroneous. We have seen that these perversions are dependent upon forces of whose action the individual himself is mainly or entirely unconscious, and that amongst those forces a prominent part is played by complexes. It has been indicated, moreover, that the perversions of evidence which occur in the sphere of pathology are due to precisely the same mechanisms, and the study of the gross and obvious effects produced in these latter cases has helped us to appreciate and understand the more restrained effects which are met with in the normal. Conscious perversion of evidence, or deliberate lying, has not been considered, although, in any complete work on the subject, a chapter on this and its psychology ought undoubtedly to be included.

Earlier in the paper we have provisionally defined rumour as the transmission of a report through a succession of witnesses, each of whom bears testimony to a statement imparted to him by his predecessor in the series. Now if this definition were sound and sufficient our task would be practically over. It would only remain to point out that at each stage of the series the testimony given would be liable to perversion in the manner we have described, and that the measure of the final perversion would be the sum of the perversions occurring at all the stages. There can be no doubt, however, that such a view would be narrow and inaccurate, and that it would fail to take into account many facts of the utmost possible importance. It is a commonplace of knowledge that a series is something more than, and different from, the mere sum of its factors. The circumstance that we are dealing with a multitude of persons, that rumour is a social and not an individual phenomenon, in itself introduces entirely new elements, and these elements must be examined and appraised. Moreover, there are other facts not in accord with our provisional definition. A rumour does not always arise as the result of a succession of reports proceeding from a single centre of origin, but sometimes appears to show a kind of spontaneous evolution, growing simultaneously from many distinct centres. Such a phenomenon suggests an origin other than a mere succession of witnesses, and reminds us of the development of myths and sagas, wherein legends almost identical in their content are evolved in widely separated nations and countries. Here again are new factors requiring investigation and analysis.

Hence, although the transmission of a report from witness to witness is an integral part of rumour, it is not the whole thereof. It is for this reason that most of the experimental work on rumour hitherto attempted has failed to produce much illumination. The experiments have been limited to the serial transmission of reports, and naturally no new facts have emerged other than those already ascertained in the investigations on evidence. Rumour is, indeed, so complex a process that experimentation is difficult to devise, and we are compelled to fall back upon the experiments provided by Nature. Fortunately she has not been niggardly during the past twenty months.

We have said that rumour is a social phenomenon, that it is something which occurs in communities, and has particular properties owing to that very fact. It is therefore necessary to take into account certain psychological principles relating to the behaviour of communities, and especially of that particular kind of community which we call a "crowd."

The psychology of the crowd has aroused much attention during recent years, and several authors have dealt with it at some length. The pioneer and the best known of these is Le Bon [4], and the last is Sir Martin Conway [6], whose "The Crowd in Peace and War" was published at the end of last year.

Le Bon pointed out that a crowd behaves differently from an individual, and he ascribed to the former an entirely distinct type of thought. His views may be shortly summarized as follows: Whoever the individuals forming a crowd may be, the fact that they have become a crowd puts them in possession of a kind of collective mind, which makes them feel, think, and act in a manner quite different from that in which each individual would feel, think, and act were he in a state of isolation. A crowd is always intellectually inferior to an isolated individual, for it does not think rationally, but is swayed by the emotion of the moment. The type of thought it exhibits is, indeed, fundamentally different from the rational thought of an individual. Crowds think in images, and each image immediately calls up a series of other images having no logical connexion with the first, but associated only by analogy or some other superficial bond. Subjective and objective are scarcely distinguished, there is no logical direction of thought, and hence contradictory ideas may be simultaneously present. For a crowd nothing is too improbable to be accepted, and a suspicion transforms itself as soon as announced into incontrovertible certainty. The convictions of crowds always assume a "religious" shape, by which term

Le Bon understands blind submission to a being supposed superior, inability to discuss dogmas, desire to spread them, and a tendency to consider as enemies all by whom they are not accepted. The dominating force responsible for all these phenomena is suggestion, to which the crowd is peculiarly and characteristically susceptible.

Conway proceeds on much the same lines, but he takes "crowd" in a rather wider sense than Le Bon, to include every "set," profession, class, or other possible congeries of people. In this sense it corresponds fairly closely to the various "social selves" of William James [11]. Conway regards the crowd as moved essentially by emotion, and contrasts this with the "reason" which is only to be found in individual thought and action.

All these observations are of great interest, both in themselves and in their bearing upon our subject, for crowds are without doubt the soil in which rumours grow and thrive, and an accurate understanding of the psychology of the crowd will probably furnish a key to at least some of the essential characters of rumour.

Much of this crowd psychology, however, is not altogether sound. The distinction drawn between the individual and the crowd is too absolute and too artificial. The doctrine that a member of a crowd exhibits intrinsically different psychological mechanisms from those he exhibits in isolation, that he becomes, as it were, a different species of animal, is crude and untrue. Crowd psychology is not intrinsically different from individual, it is simply the psychology of an individual in a particular environment, to wit, the presence of other individuals of the same species. Hence there is no more justification for the establishment of a special crowd psychology than for a "man-in-an-engineering-shop psychology" or a "man-in-a-potato-field psychology." The difference is merely one of environment, and different environments produce different reactions, whether the changes in the environment concern persons or things. The essential psychological mechanisms remain the same, although the presence of other individuals may accentuate some, and retard or inhibit others.

The fault of the crowd psychologists is not so much that they have misunderstood the psychology of the crowd, but that they have misunderstood the psychology of the individual, and have failed to appreciate that the latter shows the same type of thought as the former, though in a less obvious form. Le Bon's view that personal interest is the almost exclusive motive of an individual's conduct is a crude Benthamite doctrine which we cannot possibly accept. The statement,

again, that the conduct and thought of an individual are mainly directed by reason is opposed to the teaching of modern psychology. The emotional type of thought, which we have called "complex" thinking, holds with almost as much force in the individual as in the crowd. Genuine "rational" thinking is a comparatively rare phenomenon, and much of the thinking which we fondly imagine to be rational is in reality the result of non-conscious complexes whose action we conceal from ourselves by a process of "rationalization." The difference between individual thought and crowd thought is merely one of degree, due to the very favourable field provided in the latter for certain emotional factors which we shall afterwards define. The distinction between rational thought and "complex" thought is, of course, very real, but the line of demarcation is by no means the same as that between individual thought and crowd thought.

Among the forces responsible for "complex" thinking, a prominent place must be assigned to "herd instinct," the action of which in the psychology of civilized man has been clearly demonstrated by Trotter[17]. Herd instinct ensures that the behaviour and thought of the individual shall be in harmony with that of the community. Owing to its action the individual tends to carry out the rules of conduct which are sanctioned by the community, and to accept without question the beliefs which are current in his class. For the average man it determines his ethical code and all those opinions which are not the result of special knowledge. It must be clearly understood that herd instinct is a determining force for the major part of our individual thinking, and that it is not peculiar to so-called crowd thinking. It is a fundamental part of the psychology of each individual man, because every man is essentially a gregarious animal. Rational thinking is the only sphere in which its influence is reduced to a minimum, and genuine rational thinking constitutes but a very small part of our mental activities. It can easily be understood, however, that in a crowd the conditions are peculiarly favourable for the action of herd instinct, and that under such circumstances its influence is likely to reach a maximum. Opinions and beliefs are hence accepted more readily and with less demand for logical evidence than in the case of the isolated individual, and we should for this reason expect to find in the crowd some approximation to that entire abrogation of rationality observed by Le Bon and others. Our conclusion will therefore be that the distinction between individual thought and crowd thought is not a fundamental difference of kind, but merely one of degree. Non-rational thinking is a frequent phenomenon

in both, but it is more obvious and unrestrained in the crowd because the crowd presents conditions which are peculiarly favourable for the action of herd instinct, and herd instinct is one of the cardinal factors responsible for the non-rational type of thought.

With these conceptions at our disposal we can return to the problem of rumour, and endeavour to ascertain what relation it bears to the psychological forces at work in the crowd. This relation has been admirably analysed by Trotter [17] in his recently published "*Instincts of the Herd in Peace and War*," and the conclusions he reaches may be expressed as follows: Circumstances which stimulate herd instinct tend to arouse characteristic gregarious responses in each member of the herd. An increased sensitivity to his fellows is produced, and an increased tendency to be affected by, and to identify himself with, their alarms, hopes, opinions, and beliefs. We have just observed an example of this mechanism in the case of the crowd, where the stimulus to herd instinct provided by collecting a number of individuals together leads to a rapid contagion of non-rational opinions and decisions. It may further be stated that the strength of the gregarious responses produced are proportional to the strength of the stimulus to which herd instinct has been subjected, and that when that stimulus is maximal the gregarious responses will also attain to their maximum. Now war is probably the most intense of all possible stimuli to herd instinct, and above all a war like the present one, in which the very existence of the herd is seriously threatened. Hence we should expect under such circumstances a vast increase of all the characteristic herd manifestations, including an abnormal sensitivity to the opinions and beliefs of our fellows, and hence an abnormal prevalence and propagation of rumour. Trotter points out that war, in order to produce a maximal gregarious response, must be a really dangerous threat to the herd. The South African War, for example, was not such a threat, and correspondingly the activity and vitality of rumour were vastly less than in the present War. Again, the stimulus to herd instinct was at its maximum at the outbreak of war. One will remember the extraordinary camaraderie which then prevailed, and the changed atmosphere of the railway carriage and omnibus. Correspondingly, again, rumour was considerably more rife then than since.

When herd instinct is maximally stimulated, its action overwhelmingly dominates the mind, non-rational opinions are disseminated with prodigious ease, and the rational activities with their capacity for cool criticism are at a low ebb. In this way stories are accepted and

propagated by people who, in their more normal state, would at once detect their inherent improbability or impossibility. To what an absurd length this logic-tight process may go was well shown by one of my servants, a not unintelligent girl, who inquired breathlessly one morning whether I had heard the latest news, to wit, that "one of our airships was up last night and dropped a bomb on Ilford."

We have thus reached a position which enables us to understand the nature of the soil in which rumours grow, and the factors which make it so fertile in time of war. The next problem which confronts us is the consideration of the causes immediately responsible for the origin and development of rumours, and the classification of the different kinds of rumours which we actually find in existence. To continue the metaphor, we have to determine the nature of the seed which must be cast upon the soil, and the different species of plants which thereby develop.

With regard to the first point, the answer must evidently be that the causes leading to the origin and development of rumours are all those factors which we found, in the earlier part of this paper, to be responsible for the perversion of evidence. Chief amongst these was the action of complexes, which were shown to be capable both of perverting the report of an actual occurrence and of creating fictitious evidence in the shape of phantasy. It is clear, moreover, that the kinds of rumours produced will depend upon the nature of the complexes underlying them. We cannot attempt here to make any exhaustive classification of these kinds, but the following groups may be fairly easily distinguished:—

(1) *Rumours directly connected with the Threat to the Herd.*—These are the product of the tension and anxious expectation induced in the herd whose existence is endangered, with the resulting perversions and phantasies thereby generated. Such, for example, are rumours of invasion, of spies, of Germany's big guns, giant submarines, and submarine transports. In some of these, other subsidiary factors are also undoubtedly concerned, particularly political bias, whose supposed burial is obviously by no means so complete as we fondly endeavour to imagine. This is very clearly shown by the attitude adopted towards such rumours by the newspapers which have hauled down the old party labels in order to reappear under the new rival flags of government-baiters and government apologists. The subsidiary complexes which are thus enabled to find nourishment in this group of rumours constitute a link which carries us over to the second group.

(2) *Wish-fulfilment Rumours.* — These are produced by the mechanism which is so familiar to us in both normal and abnormal psychology, the creation of a pleasant world of phantasy in which our desires and longings are abundantly fulfilled. As examples we may mention the rumours of Zeppelins brought down in the next county, of submarines sunk in the next bay, and the most wonderful of all, the famous Russian rumour. Here again, of course, other factors play a part, some of which we shall mention subsequently.

(3) *Rumours due to Widespread and Fundamental Complexes.* — Certain complexes which belong to the essential psychological structure of every human being, and are therefore capable of being easily excited in large numbers of people, may, when an appropriate stimulus occurs, lead to the development and propagation of rumours. Such complexes tend to seize any likely material and to build phantasies upon it in which they attain some degree of satisfaction. As an example of a rumour arising in this way we may cite the war baby rumour, which seems to have owed its existence and growth to phantasies of an obviously sexual origin. In view of the eminently respectable character of many of the people who industriously propagated it, this rumour provides an interesting illustration of the indirect pathways by which the most efficiently repressed complexes will contrive to find an outlet. Rumours of atrocities, again, have probably at least one root in the sadistic and masochistic complexes which, at any rate in an undeveloped or repressed state, are more widespread than is generally thought. What may be called the instinct of cruelty seems to be an integral part of our nature, however much it may be concealed and repressed by our education and traditions. William James [11] has clearly developed this conception in his "Principles of Psychology," and he ascribes to the stimulation of this instinct the fascination which stories of atrocity have for most minds. It is easy to understand that phantasies built upon it may lead to the propagation of corresponding rumours.

Recent research has shown that fundamental and generalized complexes of the kind we are now considering, elementary complexes more or less common to the whole human race, play an important part in the development of myths and sagas, and it is interesting to note that the psychological processes concerned therein show a marked analogy with those responsible for the development of rumour [1].

There are certain peculiar aspects of rumour which merit special attention and analysis. The first of these is that curious impulse to pass on the rumour, to communicate it as soon as possible to a further

person, of which the existence is a matter of common observation. A closely similar phenomenon may be noted in the case of wit, the impulse which we all feel to pass on the last good joke we have heard [8]. We may compare with it, also, that propagandism which Le Bon has noted in crowds, the imperative desire to spread their opinions and dogmas. It is possible, without pretending that the analysis is in any way exhaustive, to indicate two groups of factors which appear to underlie this impulse.

The first group comprises the self-assertion or grandiose complexes, whose action we have already studied in the perversion of evidence. We noted there the desire to figure as a person of distinction, to occupy the centre of the stage, and to have the eyes and ears of our neighbours directed admiringly towards us; and it is clear that a similar feeling is obtaining satisfaction in the man who relates the latest rumour. It is instructive to note that the desire to achieve a position of importance in another person's mind is unquestionably present in cases of *pseudologia phantastica*, and Risch observed that his patients would only fabricate when assured of their hearer's interest. An interesting subsidiary effect of these grandiose complexes is the often observed tendency of the rumour propagator to bring the alleged events constituting the material of the rumours as closely as possible into relation with himself. Thus, when spy rumours were rife, the propagator frequently assured us that the governess with the box full of bombs had been discovered in the adjoining suburb, or in the next street, or even in the house of his cousin. The *pseudologia phantastica* patient would have said that he had himself made the discovery in his own house, but in the more normal man the powers of self-criticism are still sufficiently active to inhibit a phantasy of this degree, but not sufficiently active to prevent the minor perversions we have described. The Russian rumour provided plentiful illustrations of this process, and there were few of us who had not an aunt or a "friend in an influential position" who witnessed the travels of those wonderful troops.

The second, and probably the most important, group comprises the factors directly connected with herd instinct [17]. We have observed that this instinct, when suitably stimulated, causes the individual to seek to identify himself with the herd, and to take a part in the promotion of the herd's welfare. If this desire can obtain adequate satisfaction, the longing and unrest produced by the promptings of the instinct are at once allayed. This effect is characteristically seen in the peace of mind and freedom from worry of the man who has finally

decided to join the Army. The desire to identify oneself with the herd, to be "in it," and to play a part in its activities and strivings may easily be discerned in the propagator of rumour, and is clearly one of the factors underlying the impulse to communicate it.

Another peculiar aspect of rumour to which we desire to direct attention is the fact that it frequently exhibits a generic character—that is to say, rumour tends to assume a particular shape, which constantly appears whenever the circumstances are favourable. Thus when the Germans invaded France a rumour was immediately spread throughout Germany that the French had poisoned the wells. Similar rumours have appeared on various subsequent occasions during the present War, and have been current in former wars whenever an invasion has taken place. Of course, we are not in a position to say how much truth these assertions have contained, but their regular appearance at least arouses our suspicion. Similarly, rumours of atrocities have always tended to assume certain fixed forms. We may mention, for example, the rumour that many Belgian children were in this country whose hands had been cut off. Lastly, the best example of the generic character of rumour, whose very obviousness tends to prevent us appreciating its significance, is the circumstance that all the rumours of recent times are concerned with the War.

We are unable to present any completely satisfactory theory to account for this generic aspect, but there are certain considerations which will at least cast some light upon the question. To begin with, the fact that present-day rumours always concern the War indicates that rumours will only arise in connexion with the subject which has bound the herd together, and stimulated to their maximum degree all the forces of herd instinct. Hence in the case of the War they will tend to fall into forms which minister to the aggressive or defensive activities of that instinct, and these forms will naturally be fairly limited in number.

A further important factor in producing the generic character of rumours is the previously mentioned circumstance that the causal mechanisms fall into certain groups and thereby generate rumours of corresponding types. The last of these groups, the action of wide-spread complexes of a fundamental kind, would seem to be particularly important in this connexion. The identity of form shown by myths and sagas developed in remotely separated countries has been shown by recent research to be ascribable to their origin from complexes of primary importance which are common to the whole human race.

It is clear that this generic character in myths and sagas is closely allied to that which we are now considering in the case of rumour, and we are therefore justified in assuming that the same mechanism is probably responsible for both.

One group of rumours, those concerned with atrocities, deserves special study in this respect. It is easy to discern in some of them the action of sadistic phantasies. Stories of rape and the mutilation of women must obviously be sometimes due to this cause, and the circumstances of their origin would explain their stereotyped character. On the other hand, it is important to realize that sadistic complexes tend, not only to produce rumours of atrocities, but to express themselves in action, and to produce actual atrocities. Owing to the removal of inhibitions, always found in mobs, and certainly not least in a sacking army, we should expect these complexes to find in such circumstances an opportunity for active expression. When, in addition, the army has been bred in traditions which Mr. Trotter, in his recent book, has aptly compared with those of the wolf-pack, the opportunity for such expression is likely to be exceptionally frequent.

To this consideration we may appropriately add some concluding remarks with regard to the aims and limits of the present investigation. No attempt has been made to approach the interesting and important problem of the methods by which the perverted elements in evidence may be distinguished from those which are accurate. It is very obvious that reports are not always false, and that even rumours sometimes have a substantial basis in fact. No doubt it will ultimately be possible to devise criteria by which the wheat may be separated with certainty from the chaff, and the products of phantasy from the genuine results of observation. Law has long laboured to establish such criteria, and has evolved a procedure which is perhaps as satisfying as it is possible now to achieve. There can be no question, however, that this procedure is far from perfect, and that in it due weight is not given to factors which are obvious to the psychologist. We may reasonably expect that psychology should take a hand in the task, and provide the lawyer with information and with principles which will help him to improve the methods he now employs.

In the present paper, however, no such ambitious programme has been entertained, and the question of determining what is valid evidence and what is not has been entirely omitted. So far as rumour is concerned, the material selected has consisted solely of reports which have been subsequently admitted to be essentially false, because the problem

aimed at has been the ascertaining of the psychological mechanisms by which such false reports come into existence. How far other reports—reasonably authenticated reports of atrocities for example—are true is a question of a totally different kind, and one to be solved, not by arm-chair speculations, but by a judicial investigation.

Even with these limitations the conclusions reached can only be regarded as tentative, for the subject and its ramifications are extraordinarily complicated and involved. I can only claim to have touched the borders of a vast field, and perhaps to have suggested some likely paths along which the future explorer may attain more complete results.

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DISCUSSION.

Sir GEORGE SAVAGE: I have been much pleased and impressed by Dr. Hart's paper. It approaches the subject of rumour in what I may call a natural way. First he has considered evidence and the various causes which may strengthen or weaken it. I shall not speak on this part of the paper, but I was really delighted to find that Dr. Hart had treated rumour in a parallel to the psychology of the crowd. I have had interest in this, especially as developed in the book of Martin Conway. The contagion of feeling seen in both is eminently characteristic, and so also in both is the unreason which so generally appears. The probability of the truth in the one case, and the probability of justice being done in the other, are equally doubtful and from similar causes. The relation of untruth to developing children has always interested me, and, when asked, some years since, at a meeting which I had addressed on the evolution of the moral sense in children, how, on the evolutionary theory, I could explain the lying habit so common in children, I said that just as exuberance of vitality and muscular health led them to stand on their heads, so exuberant nervous vitality tended to overgrowth of the imagination for a time. The whole paper is so full of points of interest that time is needed to digest and utilize them.

Professor T. P. NUNN: All I have to say by way of comment on Dr. Hart's very interesting and valuable paper is that I was glad to hear him emphasize the results of Stern and others with reference to the unreliability of children's evidence. Not long ago I had occasion to be present during the police-court proceedings against a professional man charged with what the newspapers call "a serious offence." The important evidence was all given by boys aged from 9 to 11 or 12, and, taken at its face value, was distinctly incriminating. To me, however, the boys' statements seemed to show unmistakable signs of suggestion, which I had no difficulty in attributing to the well-intentioned influence of the energetic detective who had made the first inquiries into the case. The presence of suggestion was so clear in respect of the more serious of the two counts against the accused that the magistrates dismissed it. In respect of the other count the unfortunate man was committed for trial, though he was ultimately acquitted by the common-sense of a jury. My observations of this case have convinced me that persons accused of crimes of this kind may often suffer serious injustice in the absence of special precautions against the high suggestivity of children.

The PRESIDENT: I thank Dr. Bernard Hart for his very interesting paper. Rumour is defined in Webster's Dictionary as "Flying or popular report: a current story passing from one person to another without any known authority for the truth of it." But rumour is also used as a report of a fact, a story

well authorized, and the quotation given in this respect is "This rumour of *Him* went forth throughout all Judæa and the region round about." Since rumour thus used in this passage might be inferred to imply the former definition of the word, it is not surprising to find in the Revised Version of the Bible, "This report of *Him*," &c. Maudsley, in his work "Natural Causes and Supernatural Seemings," says it is hardly possible for those who have not made a study of the subject to realize how many and great events have been thought, nay seem, to happen, not because they ever did happen, but because of the strong wish and belief that they would happen. This explains the psychology of the great Russian myth to which Dr. Hart has alluded. But other sentiments play an important part, notably vanity and a desire to excite wonder and surprise in others and so to get talked about. A false report sometimes arises from erroneous observation, either because the individual is incapable of interpreting the sensations received—that is to say, there is error of perception—or because complete opportunity of observation is not available. A rumour starts by the individual telling to others a report, perhaps at first with due caution; but finding, however, that it is believed, any doubts he may have had tend to disappear, his vanity is excited, and he allows his imagination to embroider his original statement; or, if he does not himself do this, others to whom the report has been told will, for similar reasons, spread the false report, and so it passes from one person to another "without any known authority for the truth of it." It was widely believed that among the Belgian refugees in this country were children whose hands had been cut off by the Germans; that the "Huns" are capable of such frightfulness has been amply proved, and accordingly the false report, once having started, spread "without any known authority for the truth of it," as was subsequently proved by an American, who offered to build a hospital if a single case could be produced. A newspaper man came to me and said that he had heard we had a case in the hospital; I have no doubt he went fishing round in this way for copy, and thus the rumour was spread far and wide. An hysterical nurse was prosecuted for fabricating horrors which were widely believed as they found their way into the daily press. Visual hallucinations in all ages have played a dominant part in the psychology of rumour, and the vision of the angels of Mons and the controversy it has excited in the newspapers only show that human nature does not change, and "collective credulity" regarding visions still plays an important rôle of influence on the social mind.

Mr. J. C. FLUGEL: Dr. Hart has dealt in such an admirably systematic manner with his subject, and has covered so much ground in his paper, that it would seem that the chief direction in which further progress is to be made lies in that of a deeper and more searching investigation into the *precise* psychological origin and meaning of particular rumours. The actual tracing of the exact circumstances of the origin of a rumour is usually a matter of extreme difficulty. The most that can be done in many cases is a careful collecting, sifting, and criticism of available data from a comparatively small number

of individuals who have helped to give rise to or (more often) to hand on the rumour. Preliminary reports on investigations along these lines have recently been published in the *Journal of the Society for Psychical Research* as regards the "angels of Mons" and the "Russian" rumours. They seem to indicate that fully developed rumours such as these are often brought about by large and frequent distortions of memory working upon a small residuum of distorted perception in the case of a few individuals from whom the rumours presumably took their rise. Even in the absence of such investigations, however, it may sometimes be possible to obtain a deeper and fuller knowledge of the more hidden of the motive forces underlying a rumour by the application to the content of the rumour of the results of psycho-analytic work on individuals and on myths and sagas. Many of the details of rumours will then probably be found to be determined by deep-lying trends and fancies that are already familiar to students of the unconscious and its manifestations. As regards the influence of the crowd and of the gregarious tendencies upon the development of rumours, it must be borne in mind that it is seldom the influence of the crowd in the narrow sense that is at work, but rather some one or more of the more subtle far-reaching manifestations of herd and instinct to which Mr. Trotter has recently drawn attention, for it is not, as a rule, in crowds and assemblies that rumours are propagated, but rather "when two or three are gathered together" in the street, at the back door, in the smoking room, or in the train.

Mr. J. HERBERT PARSONS: I have learnt much from this extremely lucid paper. If I may offer a minor criticism, it appears to me that Dr. Hart shows a tendency to under-estimate the importance of group mentality in the natural recoil from the somewhat crude views of Le Bon and others of his school. The French school of psychologists, headed by Durkheim, have brought forward much evidence in favour of communal thought in primitive races, and it is interesting and instructive to examine the individual mind as a derivative from the group mind. I wish to emphasize the extraordinary opportunity which present conditions offer to psychologists for constructive work.

Section of Psychiatry.

President—F. W. MOTT, Major, R.A.M.C.(T.), F.R.S.

(May 30, 1916.)

The Compluetic Reaction (Wassermann) in Amentia: an Original Study of 100 Cases.

By H. F. STEPHENS.

ALL the experiments for this investigation were carried out by me in the Bacteriological Laboratories at Guy's Hospital, and my thanks are due to Dr. Eyre, the Director, for having placed every facility at my disposal, and to Dr. Ryffel, the Chemical Pathologist, for his advice and assistance. I am also indebted to my Board of Management and to my Medical Superintendent, Dr. Caldecott, for their courteous permission to undertake this investigation in the case of patients resident under their care at the Royal Earlswood Institution, at Redhill, in Surrey.

A foreword is, perhaps, necessary in explanation of the title I have assumed for this paper, a title for which I beg your kind indulgence. By the "compluetic reaction" I mean the "Wassermann reaction." It ought never to have been called the "Wassermann reaction," for, as everybody should know, and as our President has placed on record in his communication to the late Royal Commission on Venereal Diseases on this subject, not Wassermann but the Belgian Professor Bordet was the first to "discover," to study and "establish" the essential principle of the test. As England to-day is at war for justice and fair play, it is only meet and right and, perhaps, our bounden duty that the Belgian should come to his own again! The proper name for the "Wassermann reaction" is the "Bordet-Gengou phenomenon in syphilis," but, although this is rightly its proper name, such a designation, however

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romantic the association of the things discovered with the names of their discoverers may be, is nevertheless not quite correct. For it is but a mere truism to maintain that natural processes exist and are not made; they always were and will be, in spite of their discoverers; and it is with the things themselves that science is concerned. The more correct phraseology, therefore, would be "the complement-fixation or deviation phenomenon in syphilis." For this rather cumbersome expression I have taken the liberty to make the simpler term the "compluetic reaction," coining the word "compluetic" from the two words "complement" and "luetic," and as such I beg leave to submit it to your courteous and generous consideration.

This paper is divided into two sections:—

- (A) A summary of the technique employed in the experiments.
- (B) An analysis of the results obtained thereby.

(A) THE TECHNIQUE EMPLOYED.

The technique employed was that in current use at Guy's Hospital. It may be briefly summarized under the following three heads:—

- (1) The reagents used in the test.
- (2) The preliminary preparations for the test.
- (3) The test.

The Reagents.

As is well known, the reagents required for this test are:—

- (1) Antigen.
- (2) Reagin (blood serum or cerebrospinal fluid).
- (3) Complement.
- (4) Hæmolysin.
- (5) Erythrocytes.
- (6) Saline solution.

The materials used in my experiments were as follows:—

- (1) For *antigen* a 1 in 10 saline solution of an alcoholic extract of human congenital syphilitic liver.
- (2) For *reagin* only the blood sera were used. The sera were obtained from the peripheral blood of the patients. They were used undiluted.
- (3) For *complement* a 1 in 10 saline dilution of fresh guinea-pig's serum.

- (4) For *hæmolsin* a 1 in 20 saline dilution of the hæmolytic sera of rabbits immunized against human red blood corpuscles.
- (5) For *erythrocytes* a saline suspension of fresh human red blood corpuscles, obtained always independently of the bloods to be examined, and diluted 100 times (1 in 100).
- (6) A 0.9 per cent. *saline solution*, freshly made and sterile, was used for all dilutions in the test.

It may not be out of place here to note the following facts concerning the reagents:—

(1) *Antigen*.—The strength and properties of a good antigen tend to remain constant. Its characteristics must, however, be redetermined and confirmed from time to time by testing the antigen against known syphilitic reagins. This practically consists in the performance of a test in which all the factors are known except the strength and properties of the antigen. The antigen used in my experiments had been used in the routine performance of many thousands of tests, in all of which it was proved to be neither hæmolytic nor anticomplimentary, but truly antigenic in its properties.

(2) *Reagins*.—These were always tested against and compared with known "positive" and "negative" sera as "controls."

(3) *Complement*.—As is now fully recognized, the complement is the most important factor in this reaction, but unfortunately it tends to degenerate very easily. The strength of the complement therefore varies, and must be determined daily immediately before the tests are performed. The strength of the complement is best expressed in the terms of what is known as the minimal complementary dose (the "M.C.D."), the daily determination of which is one of the preliminary preparations for the test and will be described later. By means of the M.C.D. a scale of gradations can be obtained whereby what may be a purely qualitative test can be converted into a quantitative reaction. At Guy's Hospital both the qualitative and quantitative methods are in daily use. In every one of my cases, however, two minimal doses of complement were always employed, for by experiment I found that both from the qualitative and quantitative standpoints two minimal complementary doses were sufficient for each of my cases, a greater dosage resulting in the presence of an excess of complement, and, therefore, in the production of error. It is necessary to emphasize the amount of complement employed, as results with this test from the quantitative

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standpoint are only of value when expressed in terms of the M.C.D., from which it follows that, in my series of cases, even the definitely positive reactions are quantitatively very feeble.

(4) *Hæmolysin*.—The hæmolysin tends to remain constant. The strength of the hæmolysin is also best expressed in terms of what is known as the minimal hæmolytic dose (the "M.H.D."). The M.H.D. for any given brand of hæmolysin therefore tends to remain constant. The M.H.D., however, should be confirmed from time to time in the manner of the method described later.

The Preliminary Preparations.

The compluetic test, it will be remembered, is an attempt to ascertain whether a given serum in the presence of syphilitic antigen is able to "fix" complement, that is, to de-ionize it, to rob it of its oxydases, and so to render it inactive. The test is not only a means of ascertaining whether a given serum can "fix" complement, but also how much complement it can so render inactive. The test is therefore both qualitative and quantitative. It is a means of ascertaining not only the *fixation ability*, but also the *fixation capacity* of the tested serum.

Now in order to be able to "fix" complement, the given serum must contain syphilitic reagin—i.e., the syphilitic "antibody." Therefore the test becomes limited to ascertaining whether a given serum contains syphilitic reagin, and if so the quantity of syphilitic reagin it contains. Thus, in my series of cases, I was able to determine that a certain proportion of the sera examined did contain syphilitic reagin, and that the reagin so contained was invariably in small quantities, being always enough to utilize two minimal doses of complement. To effect these determinations the method employed, as will be remembered, is:—

(1) To free a given serum of any complement it will naturally contain—i.e., to "inactivate" it.

(2) To add to a measured quantity of the inactivated serum a measured quantity of antigen.

(3) To add to this mixture a measured quantity of fresh complement.

(4) A certain specified time is then allowed for these three substances to interact, and at the end of this time—i.e., usually after one hour in the air incubator at 37° C.—the presence of "free" complement is looked for by means of a delicate "colour indicator." If "free"

complement is then found to be present, the serum does not contain syphilitic reagin, and is said to be "negative." If, however, "free" complement is found to be absent (the added complement being "fixed"), the serum does contain syphilitic reagin and is said to be "positive"; the degree of its "positivity" being determined by the quantity of added complement that has been "fixed."

Such is the test. Its practical value depends upon the delicacy of the "colour indicator," whose efficiency, in its turn, is dependent upon its ability to detect the smallest traces of complement. Therefore it will be seen the preliminary preparations for the test are three in number:—

- (1) The preparation of the reagin.
- (2) The preparation of a delicate colour indicator.
- (3) The determination of the smallest amount of complement that can be detected by means of this "colour indicator."

These preliminaries will now be considered.

The Preparation of the Reagin.—In all my cases each specimen of blood for examination was taken from the peripheral circulation, about 5 c.c. of blood being withdrawn with aseptic precautions from the median basilic vein. Each specimen was allowed to clot, and the serum collected. The serum was then divided into two equal portions. One portion was heated by being placed in a hot-water bath at 56° C. for half an hour (i.e., "inactivation by heat"). The other portion was kept at room temperature for four days before being tested (i.e., "inactivation at room temperature"). The reason why each serum was divided into two portions, one being heated, the other not, is as follows: Every syphilitic serum is supposed to contain two substances, complement and syphilitic reagin (antibody); "inactivation by heat" eliminates the complement, leaving reagin for the purposes of the test. But many workers at Guy's Hospital in a long experience of many thousands of cases have found that heat also tends to damage these syphilitic reagins, some more intensely than others, so that a possibly positive serum may declare itself as negative. It has therefore been the practice at Guy's Hospital in all cases of doubtful reactions to re-test the serum, unheated, and after keeping for four days at room temperature. In my 100 cases, however, I have methodically pursued the double purpose, systematically dividing each serum into two equal portions, and regularly examining these after "inactivation by heat" and after "inactivation at room temperature." The outcome of this procedure has been that the results differed in fourteen of my 100 cases. Of these fourteen cases, four were negative heated, weakly positive

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unheated; ten were negative heated, definitely positive unheated. The results in all the other cases agreed both with the heated and the unheated portions of the sera. By testing the unheated sera in every case I was able to confirm the results obtained with the heated sera, and vice versa. Against the method of "inactivation at room temperature" it might be urged that normal sera tend to become positive on keeping, because of the factor of hydrolysis, which will naturally set free fatty and amino acids in the serum, an excess of such molecules having a decided anticomplementary action. But such a tendency would have been present in every one of the 100 sera examined, and yet in only fourteen cases did the results differ. Moreover, all these fourteen cases did not occur on the same day, under the same conditions of atmospheric pressure and temperature; and a further examination of the records showed that one occurred in a set of four, two occurred in a set of five, two occurred in a set of eight, four occurred in a set of eight, one occurred in a set of twelve, one occurred in a set of thirteen, and three occurred in a set of fourteen. So that the factor of hydrolysis does not seem wholly to explain why under the same conditions more positive results should not have occurred.

The Preparation of the "Colour Indicator."—In the preparation of a delicate "colour indicator" for this test, advantage is taken of the well-known fact that in the presence of complement and hæmolysin red blood corpuscles are destroyed, the hæmoglobin escaping to colour the medium; but that in the absence of complement no hæmolysis occurs. On the basis of this fact a mixture of hæmolysin and erythrocytes is prepared. By this mixture the hæmolysin combines with the red cells and is said to render them "sensitive" to complement. The mixture is therefore called "a suspension of sensitized erythrocytes." In the actual making of the "sensitized erythrocytes" two stages occur: (1) The determination of the minimal dose of hæmolysin that with complement will produce in a definite period complete hæmolysis of the smallest convenient quantity of red cells; (2) the mixing of the components in these proportions.

The Determination of the Minimal Hæmolytic Dose.—The reagents required for this determination are: (1) A 1 in 20 saline dilution of hæmolysin; (2) a 1 in 100 saline suspension of fresh human erythrocytes; (3) a 1 in 10 saline dilution of fresh guinea-pig's serum; (4) saline solution (0.9 per cent.). The method is as follows: A series of small test-tubes are taken, marked A, B, C, &c. Into each of these are placed

diminishing quantities of the diluted hæmolytic serum, thus: 0.10 c.c., 0.08 c.c., 0.06 c.c., &c. Then 0.05 c.c., the smallest convenient quantity, of the erythrocyte suspension is added to each tube, followed by the addition of an excess of complement, usually four minimal doses. Each tube is then filled with saline to a total volume of 1 c.c. The following example is appended as an illustration:—

	Test-tube A	Test-tube B	Test-tube C	Test-tube D
Hæmolysin (1 in 20) ...	0.10 c.c. ...	0.08 c.c. ...	0.06 c.c. ...	0.04 c.c.
Erythrocyte suspension (1 in 100) ...	0.50 „ ...	0.50 „ ...	0.50 „ ...	0.50 „
Complement (1 in 10), four doses ...	0.20 „ ...	0.20 „ ...	0.20 „ ...	0.20 „
Saline solution (0.9 per cent.) ...	0.20 „ ...	0.22 „ ...	0.24 „ ...	0.26 „
	1.00 c.c.	1.00 c.c.	1.00 c.c.	1.00 c.c.

The tubes are placed in the air incubator for one hour at 37° C. The minimal hæmolytic dose (the "M.H.D.") is the minimal amount of hæmolysin giving complete hæmolysis of 0.5 c.c. of erythrocytes after incubation in the air incubator for one hour at 37° C. In the above example the minimal amount of hæmolysin producing complete hæmolysis was found to be 0.08 c.c. (in test-tube B). Therefore the M.H.D. for the 1 in 20 saline dilution used was taken as 0.08 c.c.

The Preparation of "Sensitized Erythrocytes."—In the preparation of the "sensitized erythrocytes" used in my tests, four minimal doses of the 1 in 20 saline dilution of hæmolysin were taken for each 0.5 c.c. of the 1 in 100 suspension of red cells. These proportions were used because in actual practice an excess of hæmolysin is found to be necessary on account of the varying numbers of red cells in each 0.5 c.c. of suspension, the varying capacity of the cells for combining with hæmolysin, &c. Thus if the M.H.D. were 0.08 c.c., the recipe for the preparation would be written as follows:—

R Erythrocyte suspension (1 in 100) ...	0.5 c.c.
Hæmolysin (1 in 20), four times 0.08 c.c. (the M.H.D.) ...	0.3 „
	0.8 c.c.

Mix as much as is required in these proportions, and let the suspension stand at room temperature for fifteen minutes at least. For each dose of "sensitized erythrocytes" 0.8 c.c. of the above mixture is used.

The Determination of the Minimal Complementary Dose.—The "colour indicator" having been prepared as above, the next step is to find the smallest quantity of complement that will produce complete hæmolysis in a definite time of the estimated dose of "sensitized erythrocytes." The reagents required for this determination are:

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(1) A 1 in 10 saline dilution of the complement to be tested; (2) "sensitized erythrocytes"; (3) saline solution (0.9 per cent.). The method is as follows: A series of small test-tubes is taken, marked A, B, C, &c. Into each of these tubes diminishing quantities of the diluted complement are placed, thus: 0.09 c.c., 0.07 c.c., 0.05 c.c., &c., and then to each tube the dose of "sensitized erythrocytes" already estimated is added. Each tube is then filled with saline to a total volume of 1 c.c. The following example is appended as an illustration:—

	Test-tube A	Test-tube B	Test-tube C	Test-tube D
Complement (1 in 10) ...	0.09 c.c. ...	0.07 c.c. ...	0.05 c.c. ...	0.03 c.c.
"Sensitized erythrocytes" ...	0.80 " ...	0.80 " ...	0.80 " ...	0.80 "
Saline solution (0.9 per cent.) ...	0.11 " ...	0.13 " ...	0.15 " ...	0.17 "
	1.00 c.c.	1.00 c.c.	1.00 c.c.	1.00 c.c.

The tubes are placed in the air incubator for one hour at 37° C. The minimal complementary dose (the "M.C.D.") is the minimal amount of complement giving complete hemolysis of 0.80 c.c. of "sensitized erythrocytes" after incubation in the air incubator for one hour at 37° C. In the above example the minimal amount of complement producing complete hemolysis was found to be 0.05 c.c. (in test-tube C). Therefore the M.C.D. for the 1 in 10 saline dilution used was taken as 0.05 c.c.

The Test.

The test itself may now be described. As already stated all the quantities of the reagents used in my reactions are the same as those employed at Guy's Hospital in the routine examination of cases with the exception of the complementary doses, which did not vary in my tests, two minimal complementary doses being always used, as these were found by experiment to be both necessary and sufficient for each of my cases. The method employed was as follows:—

The Patient's Serum.—(1) For each serum two small test-tubes marked A and B were taken. (2) In each of these tubes was placed 0.1 c.c. of the patient's serum. (3) To each tube was then added two minimal doses of complement. (4) Into Tube A, but not into Tube B, was finally measured 0.1 c.c. of the antigen. Tube B was thus used as a "control" against Tube A, for as Tube B contained no antigen, one was able to note whether the patient's serum was naturally anti-complementary or not. (5) The volume of fluid in each tube was then brought to the same level by the addition, where necessary, of saline. Both tubes were then carefully shaken so that the contents of each were well mixed, the completest aseptis being observed in all the measurements.

The "Control" Sera.—Each patient's serum was always tested against and compared with two known sera, a syphilitic and a non-syphilitic serum, used as "controls." For each of these "control" sera two tubes were always prepared similar in every way to the two tubes containing the patient's serum.

For the testing of each patient's serum, then, six tubes were prepared—two for the patient's serum, two for the syphilitic serum, and two for the non-syphilitic serum. When these were ready they were placed in the air incubator for one hour at 37° C. The estimated dose of "sensitized erythrocytes" was then added to each tube, and all the tubes were replaced in the air incubator at 37° C. for another sixty minutes. They were carefully examined at regular intervals, and the results finally recorded at the end of the hour.

A note may here be added with reference to the use of "control" sera in my series of cases. All my tests were performed at the same time and on the same days on which the routine Wassermann work of Guy's Hospital was undertaken. I worked side by side with the hospital serologist, and we used the same materials for our reagents. Therefore, in addition to my own "controls," I had the advantage of the hospital's series of tubes (i.e., of those containing two minimal doses of complement) to check my reactions and to confirm the accuracy of my results.

SCHEMA OF THE TEST.

	UNKNOWN SERUM		SYPHILITIC SERUM		NON-SYPHILITIC SERUM	
	Tube A	Tube B	Tube A	Tube B	Tube A	Tube B
Reagin (undiluted) ...	0·10 c.c.	0·10 c.c.	0·10 c.c.	0·10 c.c.	0·10 c.c.	0·10 c.c.
Antigen (1 in 10) ...	0·10 c.c.	—	0·10 c.c.	—	0·10 c.c.	—
Complement (1 in 10), two doses	0·10 c.c.	0·10 c.c.	0·10 c.c.	0·10 c.c.	0·10 c.c.	0·10 c.c.
Saline solution (0·9 per cent.)	—	0·10 c.c.	—	0·10 c.c.	—	0·10 c.c.
The tubes are placed in the air incubator for one hour at 37° C.						
"Sensitized erythro- cytes")	0·80 c.c.	0·80 c.c.	0·80 c.c.	0·80 c.c.	0·80 c.c.	0·80 c.c.
	1·10 c.c.	1·10 c.c.	1·10 c.c.	1·10 c.c.	1·10 c.c.	1·10 c.c.

The tubes are replaced in the air incubator for thirty minutes at 37° C. They are then examined every ten minutes, and the results finally recorded at the end of another thirty minutes—i.e., one hour after the addition of the "sensitized erythrocytes."

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Interpretation of the Results.—The results in my series of cases were recorded as follows: (1) When no hæmolysis occurred the reaction was said to be "definitely positive." (2) When partial hæmolysis occurred the reaction was termed "weakly positive." (3) When complete hæmolysis occurred the reaction was called "negative."

Summary of the Technique.

In their preliminary report on the methods of carrying out this test, the Sub-Committee of the Section of Pathology of this Society defined what is generally understood to be "the original Wassermann test" by its essential principles as follows:—

"(1) The ingredients of the test (red corpuscles, 'antigen,' hæmolytic amboceptor, complement) are derived from *different* sources.

"(2) The serum to be tested is inactivated before use. An independent 'hæmolytic system' is employed, consisting of a suspension of red corpuscles, an inactivated hæmolytic serum, and a fresh normal serum containing complement. The hæmolytic values of the antiserum and complement are determined by a separate preliminary experiment.

"On general scientific grounds the Sub-Committee is unanimously of the opinion that, since the test is a quantitative reaction, the titre of the reagents ought, within practicable limits, to be accurately known."

From the above definition of the "original test" the technique herein described will be seen to differ, but it is sincerely hoped that the results so obtained will not therefore be very greatly impaired. Three points in connexion with this technique must be briefly recalled, inasmuch as these points are directly concerned with the interpretation of the results obtained.

First, the serum. Each serum to be tested was always inactivated before use. Each serum was divided into two equal portions; one was subjected to "inactivation by heat," the other to "inactivation at room temperature." Each portion was then tested in exactly the same way and the results carefully compared, when it was found that in only fourteen cases did the results differ. In all the other cases the results agreed both with the heated and the unheated portions of the serum. Thus by testing the unheated sera, in every case the results obtained with the heated sera were confirmed and vice versa. As some observers would maintain that more correct results are obtained by "heating" the sera, and others by testing the sera "unheated," it was hoped to avoid

the fallacies arising from both sources by systematically examining each serum in this way.

Secondly, the amount of complement used in each test. In every one of these determinations two minimal doses of complement were always employed, for by experiment it was found that two minimal complementary doses were necessary and sufficient for each of these cases, a greater dosage resulting in the presence of an excess of complement, and therefore in the production of error.

Thirdly, the "controls." Three sets of "controls" were used. (1) Each serum, whether heated or unheated, was always tested with and without "antigen," to determine whether it was naturally anti-complementary, and so to avoid if possible an excess of "positive" results. (2) Each serum, whether heated or unheated, was always tested against and compared with two known sera, a syphilitic and a non-syphilitic, under exactly the same conditions. (3) All these tests were performed at the same time and on the same days and with the same materials as the routine Wassermann work of Guy's Hospital. Therefore, in addition to the above "controls," there was always the hospital's series of tubes (i.e., of those containing two minimal doses of complement) by which to check the reactions and to confirm the accuracy of the results.

(B) THE RESULTS OBTAINED.

In this section of the paper the results obtained by the above experiments are analysed as follows:—

(1) *Total Percentage.*—Of the 100 cases examined, forty-two gave positive reactions. None of the fifty-eight "negative" cases, after a thorough clinical examination, revealed any of the characteristic lesions of syphilis, so that this group of forty-two "positive" cases would seem to include all the patients with syphilitic amentia whose blood serums were examined. Of the forty-two "positive" cases, twenty-two were "definitely positive," and twenty were "weakly positive." To many workers on this subject these figures would appear to yield a large percentage of "positive" results. The following facts, determined by a closer analysis, must therefore be stated:—

- (i) That when the sera were *inactivated by heat*, the reaction was found to be present in twenty-eight cases, being—

"Definitely positive" in	...	22	–	10	=	12 cases
"Weakly positive" in	...	20	–	4	=	16 cases
"Negative" in	...	58	+	14	=	72 cases

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- (ii) That when the sera were *inactivated at room temperature*, the reaction was found to be present in forty-two cases, being—

"Definitely positive" in	...	12 + 10 = 22 cases
"Weakly positive" in	...	16 + 4 = 20 cases
"Negative" in	...	72 - 14 = 58 cases

From which it will be seen—

- (a) That in eighty-six cases the results *agreed* both with the "heated" and the "unheated" sera, being—

"Definitely positive" in	12 cases
"Weakly positive" in	16 cases
"Negative" in	58 cases

- (b) That in fourteen cases the results *differed* both with the "heated" and the "unheated" sera, so that—

In ten cases the reaction was "negative" heated,
 "definitely positive" unheated;
 In four cases the reaction was "negative" heated,
 "weakly positive" unheated.

Now those workers who would maintain that heat tends to damage certain syphilitic reagins, some more intensely than others, so that a possibly positive serum may declare itself as negative, would regard the fourteen *differing* sera as yielding more correct results when tested after "inactivation at room temperature," and would therefore consider that a "definitely positive" reaction was obtained in twenty-two cases, a "weakly positive" reaction in twenty, and a "negative" reaction in fifty-eight, their total percentage of "positive" results being 42.

On the other hand, those observers who would urge that normal sera tend to become positive on keeping because of the factor of hydrolysis producing strong anticomplementary bodies, would consider the fourteen *differing* sera as yielding more correct results when tested after "inactivation by heat," and would therefore record them as "negative," or at best as being "doubtfully positive," and their total percentage would be 28 (16 "weakly positive," and 12 "definitely positive").

Finally, there would be some observers who would hold that because only two minimal doses of complement were used in each of these determinations, only those cases in which the results were noted as "definitely positive"—i.e., those cases in which absolutely no hæmolysis was observed—should be regarded as "truly positive." These would therefore discard the "weakly positive" and "doubtfully positive" cases as being "negative," and their total percentage of "positive" results would be 12, with which I personally am in agreement.

However the above figures may be interpreted, the point I wish to emphasize is this: that the results have been very carefully and accurately recorded; the "controls" have been numerous, strict, and efficient; and that under the conditions of the technique employed, as herein described, these results may be considered to be correct.

(2) *Sex*.—All the cases examined were males.

(3) *Age*.—The youngest of these patients was 12 years old, the five oldest were 51, 52, 52, 73 and 78 years respectively. There were fifty-seven patients between 12 and 21 years of age inclusive, twenty-one between 22 and 30 years inclusive, and seventeen between 30 and 50 years inclusive, so that the majority of the patients were boys. This fact is of some importance, for the compluetic reaction tends to vary with age, as is shown in the following tables:—

TABLE A.—SHOWING THE INCIDENCE OF THE REACTION AT DIFFERENT AGE-PERIODS.

Age-periods	Number of cases examined	Number of positive reactions at each age-period	Percentage of positive reactions to cases examined at each age-period	Percentage of positive reactions at each age-period to total number of positive reactions
Under 12 years ...	—	—	—	—
12 to 15 years inclusive	19	7	36·8	16·67
16 to 21 years inclusive	38	19	50·0	45·23
22 to 30 years inclusive	21	8	38·09	19·04
Over 30 years ...	22	8	36·36	19·04

From this table it will be seen:—

(i) That of all the positive reactions obtained twenty-six (or about 62 per cent.) occurred in patients between 12 and 21 years of age inclusive; while only eight (19 per cent.) occurred in those between 22 and 30 years inclusive, and eight (19 per cent.) in those over 30 years.

(ii) That positive reactions occurred in 45 per cent. of the patients between 12 and 21 years of age inclusive, in 38 per cent. of those between 22 and 30 years inclusive, and in 36 per cent. of those over 30 years of age.

Hence it would seem that the incidence of the reaction tended to diminish as the ages of the patients increased. But a closer study of the cases revealed an interesting modification, which also is indicated in Table A, where it is shown:—

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(i) That a larger percentage of positive reactions occurred between the ages of 16 and 21 inclusive than at any other period—viz., 45 per cent.

(ii) That 50 per cent. of the patients between the ages of 16 and 21 inclusive gave positive reactions, while about 37 per cent. of those below 16 years, and about 37 per cent. of those above 21 years, gave positive reactions.

From which it appears that the curve of the incidence of the reaction in aments tends to rise from between the ages of 12 to 15, reaching its maximum height between the ages of 16 to 21 and then gradually falling again as the ages increase.

Again, the intensity of the reaction at different age-periods is shown in Tables B and B¹ :—

TABLE B.—BEING AN ANALYSIS OF THE CASES GIVING "DEFINITELY POSITIVE" REACTIONS.

Age-periods	Total number of positive reactions at each age-period	Number of cases giving "definitely positive" reactions	Percentage of "definitely positive" reactions to the total number of positive reactions at each age-period	Percentage of "definitely positive" reactions at each age-period to the total number of "definitely positive" reactions
Under 12 years ...	—	—	—	—
12 to 15 years inclusive	7	6	85.7	27.2
16 to 21 years inclusive	19	11	57.9	47.8
22 to 30 years inclusive	8	3	37.5	13.04
Over 30 years ...	8	2	25.0	8.7

TABLE B¹.—BEING AN ANALYSIS OF THE "WEAKLY POSITIVE" CASES.

Age-periods	Total number of positive reactions at each age-period	Number of cases giving "weakly positive" reactions	Percentage of "weakly positive" reactions to the total number of positive reactions at each age-period	Percentage of "weakly positive" reactions at each age-period to the total number of "weakly positive" reactions
Under 12 years ...	—	—	—	—
12 to 15 years inclusive	7	1	14.2	5.0
16 to 21 years inclusive	19	8	42.0	42.0
22 to 30 years inclusive	8	5	62.5	26.3
Over 30 years ...	8	6	75.0	31.5

From these tables it will be seen:—

(i) That all the positive reactions, save one, occurring between the ages of 12 and 15 inclusive were "definitely positive," no hæmolysis being detected in any of the cases.

(ii) That the percentage of "definitely positive" reactions diminishes with the increasing ages of the patients.

(iii) That while there is only one "weakly positive" reaction between the ages of 12 and 15 inclusive, the percentage of such reactions increases with the increasing ages of the patients.

(iv) That if a composite graph were drawn illustrative of both these tables it would appear that the curve of the intensity of the reaction is similar to the curve of the incidence.

From this study, then, of the relationship of the incidence and intensity of the complutic reaction in aments to the age of the patient, it becomes manifest that the reaction tends to be strongest and most frequent in patients between the ages of 16 and 21, and that it appears to be more frequent and stronger in those below 16 than in those above 21 years of age; but it was not possible to determine exactly how either the incidence or the intensity curves should be drawn, the reason for this failure being shown in Table C. Such curves, however, ought to be realized when a very large number of reliable results have been tabulated and examined.

TABLE C.—SHOWING THE NUMBER OF CASES EXAMINED AND THE NUMBER OF CASES GIVING POSITIVE REACTIONS FOR EACH YEAR OF AGE FROM 12 TO 25 YEARS INCLUSIVE.

Age	C.	+	±	T.
Under 12 years	—	—	—	—
12 years old	2	—	—	—
13	2	2	—	2
14	9	3	1	4
15	6	1	—	1
16	7	1	3	4
17	8	3	1	4
18	6	1	1	2
19	7	2	—	2
20	4	—	2	2
21	6	4	1	5
22	2	—	—	—
23	6	2	—	2
24	1	—	—	—
25	2	—	1	1

C. = number of cases examined.

+

± = number of cases giving weakly positive reactions.

T. = total number of cases giving positive reactions.

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(4) *Social Status*.—The fathers of forty-two of the patients were members of the skilled trades and labourers, thirteen patients were the sons of members of the professions, and thirty were born in the mercantile classes. As these cases were chosen spontaneously and more or less haphazard, it is interesting to remark that the parents of the smallest number of these aments are members of the most highly organized occupations, viz., the professions; and it is also of importance to note that precisely those spheres of industry which have shown little or no manifestations of the social spirit contain the parents of the majority of these syphilitics (*vide* Table D).

TABLE D.—SHOWING THE SOCIAL STATUS OF THE PATIENTS EXAMINED.

Occupation of father			Number of patients examined		Number of patients giving positive reactions	
Skilled trades and labourers	42	21
Professions	13	4
Mercantile	30	14
Dead and unknown	15	3

All the 100 patients were born and bred in England (in her cities, towns, and villages) except seven. Of these seven, two were from the Channel Islands, one from the Isle of Wight, one from India, one from Barbadoes, one from Mauritius and one from Buenos Aires. The boy from Buenos Aires and the boy from the Isle of Wight gave "weakly positive" reactions; the sera of the other five were "negative."

(5) *Life-history*.—The compluetic reaction being an index of existing syphilitic infection and not in the nature of an immunity reaction, the question arises as to whether the infection in this series of cases was an intra- or an extra-uterine one—i.e., whether the syphilis was "congenital" or "acquired."

The fact that the majority of these patients came under institutional discipline and observation at a very early age, and were all of them under due protection when at home or with friends, together with the absence in them of all the physical signs and symptoms of "acquired" syphilis at any period of their lives, tends to dispose of the presence of this type of the disease in any one of these cases, the likelihood of which under the circumstances would be extremely remote. All the forty-two positive cases may therefore be said to be in varying degrees the victims of intra-uterine or "congenital" syphilis. But, save for the ophthalmoscopic and otological conditions, which unfortunately were not determined because of the obvious practical difficulties of such inquiries

in aments, the most careful examination of the patients themselves failed to reveal the presence of any of the characteristic lesions of "congenital" syphilis in any one of them, a fact which has also been noticed by Dr. Plaut, of Munich, Major Mott, and other observers.¹ Nor did a study of the records of the parental and family histories adduce evidence of the disease in any of the parents or their forbears—a fact which is less surprising, for the histories in such cases are notoriously misleading. The compluetic reaction, being the only reliable evidence in forty-two of these cases of their being syphilitic at all, was also the conclusive testimony to the presence of syphilis in either or both their parents.

A note may here be introduced on the appearance of the stigmata of "congenital" syphilis in aments. It will be remembered that "congenital" syphilis is an intra-uterine affection, and it will also be recollected that the children of syphilitic parents need not necessarily be aments. So that apart altogether from the fact that certain central nervous systems are peculiarly prone to attack by this virus, the manifestations of "congenital" syphilis would appear to be dependent upon the time of onset of the infection *in utero*, bearing a direct relation to the structural condition of the developing organism. That the majority of congenital syphilitics bear evidences of epiblastic or mesoblastic infection would seem to show that the foetus is not attacked till late in its development, or, perhaps, that the maternal defences are able to protect it till then. The presence of an earlier neuroblastic infection is manifested by the cases of juvenile general paralysis of the insane, a condition to which any of the preceding cases may succumb, while the earliest embryonic infections would appear to result either in the death of the organism or in arrested development. Thus, consider those cases of "infantilism," free from all obvious syphilitic stigmata, in which Major Mott has seen in both ovary and testis myriad colonies of the *Spirochæta pallida*. Similarly retarded development of the other members of the endocrinic glands may be found to be due to the same cause, and how closely the endocrinic system is associated with the central nervous system is daily becoming more manifest. Broadly considered, amentia may, perhaps, be interpreted as a symptom-complex of arrested brain development, such retardation being due either to an inherent inability on the part of the brain cells to grow and evolve, or to the effects of some factor inimical to their perfect fulfilment, such a factor being either traumatic, or toxic, or inflammatory in its

¹ Vide the Appendices to the Reports of the Royal Commission on Venereal Diseases, printed separately in [Cd. 7475, 1914] and [Cd. 8190, 1916].

appearance. In either case the syphilitic virus may be present as cause or coincidence—e.g., the inherent inability of the brain cells to develop may be due to some occult influence of the virus, or the results of its toxicity may be more recent and pronounced. Our knowledge, therefore, of the "stigmata" of congenital syphilis requires some replenishing. How many, for example, of the so-called stigmata of degeneracy are in reality the stigmata of syphilis? We do not know. Those of the latter, so frequently described and portrayed, are mainly somatic in incidence, originating in fully developed structures (e.g., nodes, scars, the syphilitic wig, interstitial keratitis, &c.). What is seriously needed is a more careful study of the germinal developmental stigmata (mainly microscopical in character), and the signs and symptoms with which these are associated, and by which they are rendered more evident to the naked eye—e.g., as in "infantilism," certain cases of cretinism, perhaps also certain groups of simple aments. In other words, evidences of arrested or deranged and irregular development, if shown to be directly due to the effects of the luetic virus, ought to be regarded as being in themselves "stigmata" of congenital syphilis. Again, what of the bio-chemical stigmata? One of these, surely, is that which has been studied in recent years on such a very extensive scale as "the Wassermann reaction."

To continue with this analysis, the next point to be determined was whether the syphilitic virus acting alone was responsible for the mental defect in these forty-two congenital cases, or whether it attained that end with the assistance of other factors. An analysis of such factors, based on a consideration of the so-called "causes" of amentia, is given in Table E (*see pp. 46 and 47*), from which it will be seen:—

(i) That in addition to syphilis certain reputed germinal and somatic defects are together present in seventeen cases; that these germinal defects only are present in seven cases; and the somatic defects only in eighteen cases.

(ii) That in addition to syphilis, fourteen cases have at least one of these reputed defects, fourteen others at least two such defects, and eight have three. One case has as many as six germinal and somatic defects, another has five, and four have each four of such defects. In no case is the syphilitic virus unassociated with at least one of these aetiological factors.

Other points to be noted in Table E are:—

(i) Of the forty-two positive cases the neuropathic diathesis occurs in seventeen. (Of the 100 cases examined a neuropathic inheritance

was recorded in only forty-four; so that syphilis was detected in seventeen of the forty-four cases with a neuropathic heredity. Of these seventeen cases the diathesis was recorded in seven in the parental histories only, in six in the family histories only, and in four in both family and parental histories. It appeared in seven cases on the maternal side only, in six on the paternal side only, and in four on both sides. Five of these cases gave family histories of amentia, three of insanity, and two of a "neurotic heredity." Eight of the parents were said to be "neurotic," one is insane, one had a spinal affection and was a cripple, and two died in apoplectic fits.)

(ii) There appears to be no family or parental history of alcohol.

(iii) Tuberculosis is seen to occur in the inheritance of six patients: in the family histories of four, and in the parental histories of three.

(iv) Consanguinity is present in two cases. (In one the parents were first cousins; in the other the paternal grandparents.)

(v) One of the patients, a cretin, has a paternal cousin who is likewise a cretin; and the mother and sister of another patient, also a cretin, have suffered from exophthalmic goitre. Each of these cases also has a neuropathic inheritance, but being instances of a metabolic inheritance as well they are again noted here under a separate heading.

(vi) Adverse mental states of the mother were said to have been present during the foetal development of five cases, and adverse physical conditions of the mother in three. Six others are the children of aged parents.

(vii) Fifteen are firstborn children; ten of these gave definitely positive reactions, the other five being weakly positive. It is therefore very probable that a series of abortions may have preceded the birth of most of these patients, although such a history, unfortunately, was not recorded in any case either as positive or negative evidence.

(viii) Difficult and protracted labour with instrumental delivery is recorded in seven cases (four of which were firstborn children).

(ix) One patient was a premature birth, another was a ten months' baby, and a third is the fifteenth child in his family and the second of twins.

(x) Infantile illness is cited in seventeen cases and infantile head injuries in five.

To what extent must now be considered does the syphilitic virus play its part in producing the mental deficiency in each of these cases? Its exact influence is, of course, difficult to estimate, but from a study of Table E it will be seen:—

TABLE E.—SHOWING THE FACTORS CONCERNED IN THE ETIOLOGY OF FORTY-TWO POSSIBLE CASES OF SYPHILITIC AMENTIA.

No. of case	GERMINAL DEFECTS									SOMATIC DEFECTS												
	Neuropathic inheritance			Alcoholism			Tuberculosis			Consanguinity		Aged parents		Other factors			Fetal		Natal		Infantile	
	Ancestral	Maternal	Paternal	Ancestral	Maternal	Paternal	Ancestral	Maternal	Paternal	Maternal	Paternal	Ancestral	Maternal	Paternal	Adverse mental states of the mother	Adverse physical conditions of the mother	Other factors	Firstborn	Difficult labour, &c.	Illness	Injury	
1	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	
2	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	
3	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	
4	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	
5	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	
6	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	
7	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	
8	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	
9	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	
10	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	
11	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	
12	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	
13	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	
14	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	
15	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	
16	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	
17	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	
18	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	
19	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	
20	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	
21	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	
22	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	

(A) The *Ætiological Analysis of the Twenty-two Cases giving "Definitely Positive" Reactions.*

(A) The *Etiological Analysis of the Twenty-two Cases giving "Definitely Positive" Reactions.*

(B) The Etiological Analysis of the Twenty Cases giving "Weakly Positive" Reactions.

[illegible]

a = a cretin whose paternal cousin is also a cretin.

b = a premature birth.

c = a ten months' baby.

d = the fifteenth child, and the second of twins.

e = a cretin whose mother and sister had exophthalmic goitre.

N.B.—The sera of all cases printed in *italics* were *negative* when "heated."

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(A) That the virus is undoubtedly an *auxiliary* and *augmentary* factor in the ætiology of eleven cases—viz., in Nos. 1, 2, 3, 6, 11, 23, 24, 25, 26, 28, 29.

(B) That it probably is the *exciting* or *determinant* factor in six cases—viz., in Nos. 5, 9, 10, 27, 35, 36.

(C) That it appears to be the *essential* factor in twenty-five cases—viz., in Nos. 4, 7, 8, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 30, 31, 32, 33, 34, 37, 38, 39, 40, 41 and 42.

(D) That in no case is it the *only* ætiological factor, for even on analysing Group c it will be seen:—

(a) That though in two cases (viz., 18 and 37) the syphilis appears to be wholly responsible for the amentia, it in reality has produced the mental defect by acting upon already degenerated germ plasm. Thus in No. 37 the boy's father was said to have died of phthisis; and No. 18, a Mongolian idiot, was the son of aged parents, being the eleventh child.

(b) In all the other twenty-three cases, the syphilis appears to be associated with damaged nervous tissues. Thus No. 38, a Mongolian imbecile and the second in his family, is said to have come of a healthy stock, being born under normal conditions, but two months previous to his birth his mother was shipwrecked and nearly drowned; while in the remaining twenty-two cases some adverse adventitious factor, occurring either at birth or during infancy, was always present. A study of these adventitious factors reveals the following interesting facts:—

(a) Of the fifteen firstborn children giving a positive reaction, eleven are in Group c—i.e., in the series of cases in the ætiology of which syphilis would appear to be the essential factor.

(b) Of the seven positive cases, the amental condition of each of which was said to be due to difficult and protracted labour with instrumental delivery, six are seen to be in Group c.

(c) Of the seventeen positive cases, in each of which the mental deficiency was attributed to a severe infantile illness, the syphilitic virus would appear to be the essential element in ten.

(d) While of the five positive cases in which infantile head injuries were cited as the cause of amentia, the essential ætiological factor in four would appear to be syphilis.

Obvious deductions, however, are disappointing, and, inasmuch as more than one of the above adverse adventitious factors of birth and infancy occur in many patients in Group c (as is shown in Table E), the

obvious deductions should not be drawn, nor could they be accepted as true from the results already stated. Selecting, however, for a decisive examination only those cases in which, in addition to the syphilis, there occurred *one* other adverse adventitious factor of birth or infancy, the following tentative statements may, perhaps, be presented, viz. :—

(a) That in three cases (19, 20, and 39), though the amentia was said to be due to the strain and stress of primogeniture, the mental deficiency was probably primarily and essentially the result of syphilis and not of the parturition.

(b) That syphilis was probably also the primary and essential factor in the two cases (21 and 22) in each of which the mental deficiency was directly attributed to difficult and protracted labour with instrumental delivery.

(c) That in two cases (40 and 41), perhaps also in three others (16, 17 and 31) where amentia was said to be produced by a severe infantile illness, syphilis and not the suggested illness was probably the cause of the mental defect. (The illness recorded in the histories of Nos. 31 and 41 was "infantile convulsions"; while in the other three it was due to the onset of "epilepsy," a disease from which they still suffer.)

(d) That in No. 42, in whom the mental defect was said to have appeared from the date of a head injury received in infancy, the essential cause of his amentia was probably not the injury but the syphilis, which, perhaps, was also the probable ætiological factor in No. 12, another alleged case of infantile traumatic amentia.

In conclusion, then, it should again be noted and perhaps emphasized that in none of these forty-two cases giving a positive compluetic reaction did the syphilitic virus acting alone appear to produce the amentia. In every instance it would seem that the virus was associated with and acted upon inherently defective, diseased and degenerated, or damaged nervous tissues. And this, perhaps, is only to be expected, for, as will be recollected, the children of syphilitic parents need not necessarily be aments, although, as is also known, a minority of congenital syphilitics, if untreated or ill-treated, is liable to become the victims of various nervous lesions under mental or physical strain and stress; and this is especially so—in fact, it may even be said to be only so—in those patients in whom the central nervous system is in some the least way defective. Given a healthy nervous system, congenital syphilis, as is proved by common medical experience, will not produce amentia. On the other hand, it is quite possible, and even reasonable,

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to suppose that given an exceedingly virulent neurotoxic type of syphilis *in utero* the healthiest nervous system would be likely to fall a victim to the virus. Between these two extremes, the various grades of syphilitic amentia may be found.

(6) *Classification*.—Mr. Tredgold, in his book "Mental Deficiency," maintains that "there are two fundamentally different forms of amentia; there are also innumerable degrees; and it is convenient to describe certain distinctive clinical varieties." The forms of amentia Mr. Tredgold has called primary and secondary; the degrees are now jumbled together into four groups by Act of Parliament; the clinical varieties are well known. In the following paragraphs the lines of classification suggested by Mr. Tredgold will be followed with slight modifications:—

(a) The forms of amentia: Of the 100 cases serologically examined, seventy-six were cases of primary amentia and twenty-four cases of secondary amentia. Of the forty-two cases giving a positive compluetic reaction, thirty-two were cases of primary amentia, ten cases of secondary amentia, so that of all the cases of primary amentia examined, a little over 42 per cent. were positive, and of all the cases of secondary amentia examined, nearly 42 per cent. were positive. Moreover, as to the intensity of the reaction in each group, nine of the thirty-two cases of primary amentia gave "definitely positive" reactions, thirteen "weakly positive" reactions, and ten "doubtfully positive" reactions; while three of the cases of secondary amentia gave "definitely positive" reactions, three "weakly positive" reactions, and the remaining four "doubtfully positive" reactions.

(b) The degrees of amentia: By Act of Parliament the "innumerable degrees" of amentia are now legally grouped as four—idiots, imbeciles, feeble-minded, and moral imbeciles. All the 100 patients examined were either idiots or imbeciles. As a matter of fact, seventy of them may be considered to be imbeciles and thirty are idiots. Half of these thirty idiots gave positive reactions, in five the reaction being "definitely positive," in five "weakly positive," and in five "doubtfully positive"; while of the seventy imbeciles, twenty-seven gave a positive reaction, and of these seven were "definitely positive," eleven "weakly positive," and nine "doubtfully positive."

(c) The clinical varieties of amentia: No distinctive clinical type of syphilitic amentia has been described. Of the known clinical varieties the following occurred among the 100 cases examined:—

(I) PRIMARY AMENTIA.

(1) Microcephalus	Nil
(2) Mongolianism	11 cases
(3) Simple amentia	65 cases

(IA) PRIMARY AMENTIA WITH COMPLICATIONS.

(1) With paralysis only	2 cases
(One mongol and one simple primary ament.)						
(2) With convulsions only	26 cases
(All simple primary aments.)						
(3) With paralysis and convulsions	Nil

(II) SECONDARY AMENTIA.

(1) Hydrocephalus	3 cases
(2) Hypertrophicism	1 case
(3) Cretinism	4 cases
(4) Simple amentia	16 cases

(IIA) SECONDARY AMENTIA WITH COMPLICATIONS.

(1) With paralysis only	Nil
(2) With convulsions only	10 cases
(One hydrocephalic, one hypertrophic, and eight simple secondary aments.)						
(3) With paralysis and convulsions	5 cases
(One cretin, two hydrocephalic, and two simple secondary aments.)						

Each of these varieties will now be considered (*vide* also Table F).

(1) *Microcephalus*.—Unfortunately no case was examined.

(2) *Mongolianism*.—Of the eleven cases examined only three were positive. In other words, in over 72 per cent. of these cases the sera did not react to the test. Of the three mongols giving a positive reaction two were doubtfully positive and the other was weakly positive. Brief records of the life-histories of these eleven mongols are appended:—

The Negative Cases.

Case 1.—Idiot, aged 14. (1) Ancestral history, *nil*. (2) Parental history, *nil*. Father a farm labourer. (3) Personal history: The third of five children; birth normal; infancy and childhood healthy.

Case 2.—Idiot, aged 25. (1) *Nil*. (2) *Nil*. Father a labourer. (3) The seventh of seven; fright of shipwreck to mother when three months pregnant; an eight months' child; birth normal; infancy and childhood healthy.

Case 3.—Idiot, aged 20. (1) *Nil*. (2) Father somewhat dull; father and mother aged about 35 when patient was born; father a clerk. (3) The second of three; fall of mother at seventh month; birth normal; infancy and childhood healthy.

Case 4.—Imbecile, aged 26. (1) *Nil*. (2) Father died of nephritis; mother healthy. (3) The fifth of six; fall of mother just before full term; birth normal; infancy and childhood healthy.

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Case 5.—Imbecile, aged 20. (1) Paternal grandmother, aunt and sister epileptic. (2) *Nil.* Father manager of brickfields. (3) The only child; birth normal; infancy and childhood healthy.

Case 6.—Imbecile, aged 18. (1) *Nil.* (2) *Nil.* Father an engineer fitter. (3) The fifth of eight; birth normal; infancy and childhood healthy.

Case 7.—Imbecile, aged 22. (1) *Nil.* (2) Mother always very delicate; father a policeman. (3) The eighth of nine; birth normal; infancy and childhood healthy; has right internal strabismus.

Case 8.—Imbecile, aged 23. (1) *Nil.* (2) *Nil.* Father a bank clerk. (3) The fourth of five; a seven and half months' child; birth normal; infancy and childhood moderately healthy.

The Positive Cases.

Case 9.—Imbecile, aged 21. (1) *Nil.* (2) *Nil.* (3) The second of four; fright of shipwreck and drowning to mother two months before his birth; birth normal; infancy and childhood healthy; C.R. weakly positive.

Case 10.—Imbecile, aged 17. (1) *Nil.* (2) *Nil.* Father a doctor. (3) The firstborn; birth normal; infancy and childhood healthy; C.R. doubtfully positive.

Case 11.—Imbecile, aged 27. (1) *Nil.* (2) *Nil.* Father a furniture remover (died from accident). (3) Eleventh of eleven; birth normal; infancy and childhood fairly healthy; C.R. doubtfully positive.

(3) *Hydrocephalus.*—Only three cases were examined, and one of these gave a doubtfully positive reaction. Their life-histories are briefly as follows:—

Case 1.—Imbecile, aged 15. (1) *Nil.* (2) *Nil.* Father a labourer. (3) The sixth of ten; protracted labour and instrumental delivery; left hemiplegia, Jacksonian epilepsy; C.R. doubtfully positive.

Case 2.—Imbecile, aged 27. (1) *Nil.* (2) Mother died of erysipelas; father a doctor. (3) The second of seven; birth normal; infantile convulsions; C.R. negative.

Case 3.—Imbecile, aged 40. (1) *Nil.* (2) Father died of phthisis; mother died of "cancer"; father a schoolmaster. (3) The eighth of ten; worry to mother during pregnancy; birth normal; epilepsy when young; has left internal strabismus; C.R. negative.

(4) *Hypertrophicism.*—One case was examined, giving a definitely positive reaction. This boy is aged 13, and the fourth child in a family of four. When he was born his father was aged 70 and his mother aged 40. No consanguinity existed between his parents; no phthisis, alcoholism, or insanity was said to be present in the life-histories. His

father died of general peritonitis; his mother is alive and healthy. Instrumental delivery was necessary at his birth, but with the exception of recurrent convulsive attacks from which he at present suffers, he has always been in good health. Mentally, he is an imbecile of a cheerful temperament and a happy disposition.

(5) *Cretinism*.—Four cretins were examined, and of these two gave positive reactions, one being definitely positive and the other doubtfully positive.

Case 1 (giving a definitely positive reaction).—A boy, aged 16, and the second of a family of six, all of whom are said to be normal. A paternal cousin is a cretin; the mother is said to be "neurotic"; otherwise the life-histories are normal. Both his parents are healthy, his father being a fisherman. The mother attributes the patient's condition to the fright she sustained on seeing the cretin cousin for the first time while pregnant with the patient. His birth was normal. He has taken extracts of the thyroid gland since the age of 18 months. He was operated on as a child for cerebral abscess. At the present day he is a strong, healthy, robust boy. He suffers occasionally from epistaxis, and recently he had two attacks of melæna. Mentally he is imbecile.

Case 2 (giving a doubtfully positive reaction).—A boy, aged 16, and the second of a family of three. No defect is recorded as present in his ancestral history. Both his parents are "neurotic," but physically in good health. His father is a warehouseman. His mother and her eldest child, his sister, have had exophthalmic goitre. The birth of the patient was normal. He has had thyroid from an early age. He is and has always been in good health. Mentally he is imbecile.

Case 3 (giving a negative reaction).—An idiot, aged 16, with a history of "neurotic heredity." His father, a parson, died of "heart disease"; his mother is alive and healthy. He is the only child, and instrumental delivery was necessary at his birth. He has had thyroid from an early age. He is epileptic. He also has a left otorrhœa, a right ptosis, and undescended testes.

Case 4 (giving a negative reaction).—An imbecile, aged 22, and the second in a family of five. Recorded in his family history is the fact that a maternal niece is feeble-minded and subject to epilepsy. His father died of "cancer of the kidneys." His mother is in good health, but she says she was unable to speak distinctly till the age of 12. His eldest sister is a cretin. His birth was normal, but his "mother used to think a great deal about a cretin when carrying him." No infantile injury or other illness is recorded. He has had thyroid from the age of 2, and is to-day robust, strong, and in good health. He is very deaf.

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(6) *Simple Amentia*.—In the book aforementioned, Mr. Tredgold writes: "The majority of persons suffering from primary amentia present no special distinguishing features other than the anatomical and physiological anomalies common to aments in general; they may therefore be termed simple aments, and they correspond to the 'genetous' group of Ireland." "This term," he adds in a note, "is open to the objection that all primary aments may in reality be called 'genetous.'" In this paper both the "genetous" group of Ireland and the "simple aments" of Mr. Tredgold are called by the more exact name of "simple primary amentia." It is here suggested that the

TABLE F.—SHOWING THE RELATION OF THE REACTION TO THE CLINICAL VARIETIES OF AMENTIA EXAMINED.

Clinical variety	Number of cases	Number of "definitely positive" reactions	Number of "weakly positive" reactions	Number of "doubtfully positive" reactions	Number of "negative" reactions
(A) <i>Primary amentia</i> :—					
(1) Microcephalus ...	—	—	—	—	—
(2) Mongolianism ...	11	—	1	2	8
(3) Simple amentia ...	65	10 (15·3 per cent.)	11 (17 per cent.)	8 (12·3 per cent.)	36 (72 per cent.)
(B) <i>Secondary amentia</i> :—					
(1) Hydrocephalus ...	3	—	—	1	2
(2) Hypertrophism ...	1	1	—	—	—
(3) Cretinism ...	4	1	—	1	2
(4) Simple amentia ...	16	—	5	1	10
(C) <i>Amentia with complications</i> :—					
(1) Amentia with paralysis ...	7	—	—	2	5
(2) Amentia with convulsions ...	41	5 (12·2 per cent.)	9 (22 per cent.)	7 (17 per cent.)	20 (48·8 per cent.)

term "simple amentia" should include a larger number of cases than those concerned in Mr. Tredgold's definition; that so it should be capable of division into two classes, corresponding to the two forms of amentia, and that these two classes of simple amentia should be termed "simple primary amentia" and "simple secondary amentia" respectively. Mr. Tredgold's definition, in fact, may be adapted as follows: Persons suffering from simple amentia present no special distinguishing features other than the anatomical and physiological anomalies common to aments in general; they may be divided into two classes corresponding to the two forms of amentia and may be termed

"simple primary aments" and "simple secondary aments" respectively. By simple primary amentia would be understood the "simple amentia" described by Mr. Tredgold, while simple secondary amentia would constitute all those cases of secondary amentia in which the mental defect is due to gross cerebral lesions that are the results of toxic, inflammatory, or vascular causes, but the patients themselves are not distinguished by any of the special characteristics peculiar to the clinical varieties of secondary amentia definitely known and described (*viz.*, hydrocephalus, cretinism, &c.).

Each of these two groups of simple amentia will now be considered.

(a) Simple primary amentia: Of the 100 cases examined, sixty-five were cases of simple primary amentia, and of these twenty-nine (or 44.6 per cent.) gave positive reactions. Moreover, of these sixty-five cases, twenty-seven were cases of amentia with complications, and thirty-eight were uncomplicated cases. Of the latter, fourteen (or 36.8 per cent.) gave positive reactions, while of the former fifteen (or 55.6 per cent.) gave positive reactions. Again, of the twenty-nine cases giving positive reactions, ten gave "definitely positive" reactions, eleven "weakly positive" reactions, and eight "doubtfully positive" reactions. Of all the "definitely positive" reactions, 40 per cent. were given by the complicated cases, and of all the "weakly positive" reactions 45 per cent. were given by the uncomplicated cases.

(b) Simple secondary amentia: Sixteen of these cases were examined. *Ætiologically*, they may be classified as follows: Simple secondary amentia due to—(i) "infantile convulsions," seven cases; (ii) recurrent convulsions in early childhood, three cases; (iii) other illnesses in infancy, four cases; and (iv) head injuries in infancy, two cases. The first two of these groups will be considered in the section dealing with "amentia with convulsions." It may here be recollected that the *ætiological* group of simple secondary amentia due to "recurrent convulsions in early childhood" was known by the older writers as "eclampsic amentia." To-day Mr. Tredgold calls the same group of cases "epileptic amentia," distinguishing them from "amentia with epilepsy," where the convulsions are a complication and not the cause of the amentia. Both these terms, however, tend to confusion and are therefore not employed in this paper. Of the remaining two *ætiological* groups of simple secondary amentia, the sera of the four cases due to "other illnesses in infancy" gave negative reactions, the illnesses recorded being "sunstroke," "meningitis," "illness at teething," and "mastoiditis." Both the cases due to head injuries in infancy gave

positive reactions, one being doubtfully positive and the other weakly positive. In the aetiological Table E these two cases are shown as Nos. 12 and 42 respectively, and they are very probably cases in which the syphilitic virus and not the alleged injury is the primary and essential cause of the mental defect.

(7) *Amentia with Complications.*—Of the 100 cases examined forty-three were cases of amentia with complications. Of these forty-three cases twenty-one (or 48·8 per cent.) gave positive reactions, while of the fifty-seven uncomplicated cases twenty-one (or 36·8 per cent.) gave positive reactions; so that of all the positive reactions obtained half were given by the uncomplicated cases and half by the complicated cases. Again of the seventy-six cases of primary amentia examined, twenty-eight (or 36·8 per cent.) were cases with complications, while fifteen of the twenty-four cases of secondary amentia (i.e., 62·5 per cent.) were complicated cases. Of all the cases of primary amentia with complications, fifteen (or 53·5 per cent.) gave positive reactions, while of the uncomplicated cases of primary amentia seventeen (or 35·4 per cent.) gave positive reactions. Of all the cases of secondary amentia with complications six (or 40 per cent.) gave positive reactions, while of the uncomplicated cases of secondary amentia four (or 44·5 per cent.) gave positive reactions.

The cases of amentia with complications will now be considered under two heads: (a) Cases with paralysis; (b) cases with convulsions.

(a) *Amentia with paralysis:* The physical condition of seven of the 100 patients was complicated with paralysis, the types of paralysis being as follows: Hemiplegia, three cases; strabismus, three cases; and ptosis, one case. Of these seven cases, two gave doubtfully positive reactions, both being hemiplegias and cases of secondary amentia. One of these two, a hydrocephalic, is also subject to "Jacksonian epilepsy."

(b) *Amentia with convulsions:* At the present day seventeen of the 100 cases examined suffer from recurrent convulsive attacks commonly designated by the convenient group-name of "epilepsy." In addition to these, eleven others have suffered from similar attacks when younger, but are not so afflicted now; and twelve others are said to have had "infantile convulsions." Moreover, one other case is subject to "Jacksonian epilepsy." In all, then, forty-one of the 100 cases examined have had convulsive attacks at some period of their lives. These cases are analysed in Table G.

TABLE G.—BEING AN ANALYSIS OF THE FORTY-ONE CASES OF AMENTIA WITH CONVULSIONS.

Clinical variety	Number of cases	Number of "definitely positive" reactions	Number of "weakly positive" reactions	Number of "doubtfully positive" reactions	Number of "negative" reactions
<i>Recurrent convulsions at present day :—</i>					
(1) Simple primary aments	15	2 (13 per cent.)	4 (27 per cent.)	3 (20 per cent.)	6 (40 per cent.)
(2) Hypertrophic ...	1	1	—	—	—
(3) Cretin (with paralysis)...	1	—	—	—	1
<i>Recurrent convulsions in childhood :—</i>					
(1) Simple primary aments	7	2	1	2	2
(2) Hydrocephalic (with paralysis)	1	—	—	—	1
(3) Simple secondary aments	2	—	—	1	1
(4) Simple secondary aments (with paralysis)	1	—	—	—	1
<i>"Infantile convulsions" :—</i>					
(1) Simple primary aments	4	—	1	—	3
(2) Hydrocephalic ...	1	—	—	—	1
(3) Simple secondary aments	6	—	3	—	3
(4) Simple secondary aments (with paralysis)	1	—	—	—	1
<i>"Jacksonian epilepsy" :—</i>					
(1) Hydrocephalic (with paralysis)	1	—	—	1	—

From this table it will be seen :—

(i) That, excluding the "Jacksonian epileptic," of the forty remaining cases of amentia with convulsions, twenty (i.e., 50 per cent.) gave positive reactions, five being "definitely positive," nine "weakly positive," and six "doubtfully positive."

(ii) That excluding also the cases of "infantile convulsions" there are twenty-eight patients who are or have been subject to recurrent convulsive attacks. Of these, sixteen (a little over 57 per cent.) gave positive reactions, five being "definitely positive," five "weakly positive," and six "doubtfully positive."

(iii) That of the seventeen cases suffering at the present day from recurring convulsive attacks, commonly designated by the group-name of "epilepsy," ten, or nearly 59 per cent., gave positive reactions; and of these positive reactions 30 per cent. were "definitely positive."

Hence it would seem from the above study that of all the patients who have had convulsive attacks at some period of their lives, over 50 per cent. gave positive reactions; while of those who might have

been designated "epileptic" at some period of their lives, a little over 57 per cent. gave positive reactions; and of those diagnosed as "epileptic" to-day, nearly 59 per cent. gave positive reactions.

Again comparing the figures given in Table G with the total number of cases giving positive reactions (viz., forty-two cases), it will be seen that of all the patients giving positive reactions twenty-one (or 50 per cent.) have had convulsive attacks at some period of their lives; sixteen (or 38 per cent.) might have been designated "epileptic" at some period of their lives; and ten (or nearly 24 per cent.) are diagnosed as "epileptic" to-day.

Considering now the non-convulsive cases, it will be remembered that fifty-nine of the 100 cases examined have never had a convulsive attack of any kind at any period of their lives. Of these fifty-nine cases twenty-one gave positive reactions. In other words, half the total number of positive reactions in this series occurred in non-convulsive cases, and the other half in those who have had convulsive attacks at some period of their lives. To be more exact, 50 per cent. of the positive reactions occurred in non-"epileptic" cases and 38 per cent. in those cases which might have been designated "epileptic" at some period of their lives. On the other hand, as already noted, a little over 57 per cent. of these "epileptic" cases gave positive reactions, while of the non-"epileptic" cases only 35.5 per cent. gave positive reactions.

Other facts to be noted are as follows:—

(a) Seventy-six cases of primary amentia were examined, and of these twenty-six were cases with convulsions. Of the convulsive cases, fifteen (or 57.7 per cent.) gave positive reactions, while of the fifty non-convulsive cases seventeen (or 34 per cent.) gave positive reactions.

(b) Fifteen of the twenty-four cases of secondary amentia were cases with convulsions. Of these convulsive cases six (or 40 per cent.) gave positive reactions, while of the non-convulsive cases four (or 44.5 per cent.) gave positive reactions. Moreover, of the fifteen convulsive cases ten were cases with convulsions only, and of these five (or 50 per cent.) gave positive reactions.

(c) Eighty-one cases of simple amentia were examined, and of these thirty-six were cases with convulsions. Of the convulsive cases nineteen (or 52.7 per cent.) gave positive reactions, while of the forty-five non-convulsive cases sixteen (or 35.5 per cent.) gave positive reactions. Moreover, thirty-four of these cases were cases with convulsions only, and nineteen (or 55.8 per cent.) gave positive reactions.

Again, of the thirty-six cases of simple amentia with convulsions, twenty-six were cases of primary amentia and ten of secondary amentia. Of the twenty-six cases of simple primary amentia with convulsions, fifteen (or 57·6 per cent.) gave positive reactions, while of the non-convulsive cases of this type fourteen (or 35·8 per cent.) gave positive reactions. Of the ten cases of simple secondary amentia with convulsions, four (or 40 per cent.) gave positive reactions; of the eight cases of this type with convulsions only, four (or 50 per cent.) gave positive reactions; while of the non-convulsive cases two (or 33·3 per cent.) gave positive reactions.

The last point to be investigated was the relation of the intensity of the "epilepsy" and the frequency of the convulsive attacks to the incidence and intensity of the reaction. Unfortunately no such relations were obtained. Three of the "negative" cases, for instance, were the severest cases of "epilepsy" examined; while of the "positive" cases the most severe and the mildest gave "definitely positive" reactions, the serum of the second worst was only "weakly positive," and so on. One slight feature of this series of cases, however, should be noted. It was found that the convulsive attacks in the "positive" cases tend to be far more frequent during the night and the early hours of the morning than they are during the day, while in the "negative" cases they are most frequent during the day. The intensity of the convulsive attacks in each instance appears to be characterized also in the same way. This feature of the "positive" cases, therefore, tends to bring them into line with other syphilitic conditions—e.g., the syphilitic headache, which is said to be most intense towards the early hours of the morning.

RECAPITULATION.

The main points of this paper may be recapitulated as follows:—

(1) *The Incidence of the Compluetic Reaction in the Cases of Amentia Examined.*—The blood sera of 100 aments were examined, and a "positive" reaction was found to be present in forty-two; but only twelve of these are to be regarded as "truly positive."

(2) *The Intensity of the Reaction in the Cases of Amentia Examined.*

(i) Sera were tested heated and unheated. Sera in which no hæmolysis occurred were recorded as "definitely positive"; those in which partial hæmolysis occurred as "weakly positive." Sera in which the results differed when heated and unheated were recorded as "doubtfully positive." Of the forty-two sera giving a positive reaction,

in twelve the reaction was found to be "definitely positive," in sixteen "weakly positive," and in fourteen "doubtfully positive"; but only the twelve "definitely positive" reactions are to be regarded as "truly positive."

(ii) Quantitatively, even the "definitely positive" reactions are to be considered as feeble reactions, for the syphilitic reagin present in each of these cases was invariably found to be in small quantities, being always enough to utilize two minimal doses of complement.

(3) *The Relation of the Presence of the Reaction to the Sex of the Patients Examined.*—This was not determined, as all the aments examined were males.

(4) *The Relation of the Presence of the Reaction to the Age of the Patients Examined.*—The reaction was found to vary with age. As a rule, it tended to diminish in incidence and intensity as the ages of the patients increased. It appeared, however, to be strongest and most frequent in patients between 16 and 21 years of age; and it tended to be more frequent and stronger in those below 16 than in those above 21 years of age. In considering the total percentage of the positive results obtained, it should be remembered that the majority of the patients examined were boys, 38 per cent. being between 16 and 21, and 57 per cent. under 21 years of age.

(5) *The Relation of the Reaction to the Forms of Amentia Examined.*—The reaction was obtained in a little over 42 per cent. of the cases of primary amentia, and in nearly 42 per cent. of the cases of secondary amentia. It tended to be a little stronger in the latter group of cases.

(6) *The Relation of the Reaction to the Degrees of Amentia Examined.*—Only idiots and imbeciles were examined. The reaction appeared to be more frequent and stronger in the idiots than in the imbeciles; thus, 50 per cent. of the idiots examined gave a positive reaction, and in 16.6 per cent. the reaction was found to be "definitely positive," whereas it was present in 38.5 per cent. of the imbeciles, being "definitely positive" in 10 per cent.

(7) *The Relation of the Reaction to the Clinical Varieties of Amentia Examined.*

(i) In over 72 per cent. of mongols the serum did not react to the test. Of the eleven cases examined, a positive reaction was obtained in three, being "weakly positive" in one and "doubtfully positive" in the other two.

(ii) One of three cases of hydrocephalus gave a "doubtfully positive" reaction.

(iii) Only one case of hypertrophicism was examined, and this was found to be "definitely positive."

(iv) A positive reaction was obtained in two of four cretins, being "definitely positive" in one, and "doubtfully positive" in the other.

(v) Eighty-one cases of simple amentia were examined, and the reaction was found to be "definitely positive" in ten, "weakly positive" in sixteen, and "doubtfully positive" in nine. Excluding the "doubtfully positive" cases the reaction was found to be present in 32 per cent. of simple aments. It appeared to be more frequent in the cases of simple primary amentia than in the cases of simple secondary amentia.

(vi) Of the seven cases of amentia with paralysis, two, both hemiplegias, gave "doubtfully positive" reactions.

(vii) Excluding the "doubtfully positive" cases, the reaction was present in 41.2 per cent. of patients suffering at the present day from recurring convulsive attacks, commonly designated by the group-name of "epilepsy"; in 35.7 per cent. of those diagnosed as "epileptic" at some period of their lives; and in 23.7 per cent. of non-"epileptic" cases. It should also be noted that of the simple primary aments with recurrent convulsions to-day, a "definitely positive" reaction was obtained in only 13 per cent.

(8) *The Rôle of Syphilis in the Aetiology of the Cases of Amentia Examined.*—Three points are to be noted:—

(i) That as the compluetic reaction is an index of existing syphilis, all the positive cases are to be regarded as weak syphilitic infections.

(ii) That these weak infections were intra-uterine or "congenital" in origin.

(iii) That the syphilitic virus did not appear to be wholly responsible for the amentia in each of these cases, but seemed rather to have been associated with and to have acted upon inherently defective, diseased and degenerated, or damaged tissues.

In conclusion, I should like to state how very grateful I am to Sir George Savage for his kindly interest in and generous appreciation of this my work, whereby it was submitted to the authoritative criticism of Major Mott, who, though he regretted that the technique employed was not in strict conformity with that of the "original" test, yet agreed the results obtained were of sufficient value to be placed before this meeting.

APPENDIX.

Showing some of the results published.

Names of observers	Number of cases examined	Number of "positive" results obtained	Percentage of "positive" results obtained
<i>In France.</i>			
Raviart, Breton, &c. [1] ...	246	76	30.8
<i>In Denmark.</i>			
(a) Lippmann [2] ...	78	7	8.9
(b) <i>Idem</i> ...	(?)	(?)	13.2
<i>In Germany.</i>			
Kellner, Clemens, &c. [3] ...	216	8	3.7
(a) Dean [4] ...	(?)	(?)	12.0
(b) <i>Idem</i> ...	330	51	15.4
Thomsen, Boas, &c. [5] ...	2,061	31	1.5
Kröber [6] ...	262	56	21.3
<i>In America.</i>			
Atwood [7] ...	204	30	14.6
W. C. Stoner and E. L. Keiser [8] ...	1,050	83	7.9
Dawson [9] ...	—	—	4.0
Stevens [10] ...	21	2	9.5
<i>In England.</i>			
Muirhead [11] ...	5	—	—
Schölberg and Goodall [12] ...	46	15	32.6
Chislett [13] ...	22	11	50.0
Gordon [14] ...	400	66	16.5
Fraser and Watson [15] ...	205	123	60.0
(a) Rees Thomas [16] ...	163	8	4.9
(b) <i>Idem</i> ...	—	—	10.0
Robertson and Findlay [17] ...	15	(?) 9	59.0
F. E. Batten [18] ...	2	—	—
Mott [19] ...	257	21	8.1
H. F. Stephens ...	100	12	12.0

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- [19] MOTT. "Final Report of the Royal Commission on Venereal Diseases" (Cd. 8189, 1916), Appendices, p. 144.

DISCUSSION.

Dr. PERCY SMITH: Before the Wassermann test was discovered the percentage of definite syphilitic cases in mental defectives (idiots and imbeciles) appeared to be only 2 or 3 per cent., and the enormous difference in the percentages now shown by various observers raises doubt as to the real proportion of syphilitic cases. It is interesting that in the case of "mongol" idiots the percentage of syphilitics appears to be low, these cases being commonly the youngest child of a large family when the mother is approaching the limit of reproductive function.

Dr. SHUTTLEWORTH: In reference to the statistics of syphilis and mental defect quoted by Dr. Percy Smith, these were published by Dr. Fletcher Beach and myself in Hack Tuke's "Dictionary of Psychological Medicine" so long ago as 1892. They were based upon clinical signs and family histories of 2,380 cases investigated at the Darenth and Royal Albert Asylums, and gave a very low estimate (under 2 per cent.) of the influence of inherited syphilis as a factor of idiocy, &c. The late Dr. Langdon-Down also stated that in his large experience of idiots at Earlswood, as well as from pathological investigations as Physician to the London Hospital, he was of opinion that not more than 2 per cent. were the subjects of congenital syphilis. The *Spirochæta pallida* was, however, not discovered till 1905, and the earlier statistics rested solely on personal stigmata and family history. Subsequently the Wassermann reaction came into vogue as a test, and very varying reports have been issued by successive observers. In the new edition (just published) of "Mentally Deficient Children," there appears a table of percentages obtained by fourteen different observers, varying from 1.5 to 60, in which positive reactions to the Wassermann test have been recorded in the blood of congenital cases of mental defect. Dr. Stephens apparently found such reaction in 42 per cent. of his 100 cases at Earlswood. Unfortunately I was prevented from arriving in time to hear the early part of the paper, in which presumably Dr. Stephens has described the technique he has adopted. But the discrepancies noted lead to the suspicion that the methods used by the various observers cannot have been uniform, and consequently the results must be to some extent discounted. The inquiry is a most important one, and I trust that Dr. Stephens will utilize the material at Earlswood to the full. I am specially interested in the comparison made by Dr. Stephens as to reaction in certain characteristic types. The mongolian type, for instance, has shown comparatively little "compluetic" reaction, and this accords with clinical experience that such cases are "exhaustion products" rather than of toxic

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origin, though in exceptional cases a syphilitic taint may give rise to maternal exhaustion. I have always suspected that certain hydrocephalic cases are of syphilitic origin, and that is a point on which the compluetic reaction will shed scientific elucidation.

Sir GEORGE H. SAVAGE: I regret the smallness of the meeting, for I think the paper by Dr. Stephens one of the most important which have been read at the Section. It will be fully appreciated when it is in print. A very great change has taken place in the opinion of doctors in relationship to syphilis as a possible or partial cause of mental deficiency. Recently a paper has been read at the Medico-Psychological Society on the influence of toxins in producing mental deficiency. We all allow that syphilis is the most dangerous toxin to the nervous systems, and we are prepared to recognize it as a cause of amentia. The former authorities on idiocy, such as Langdon-Down and Ireland, did not recognize it as a factor. The great difference between the percentage of positive Wassermann reactions met with at Earlswood from similar observations in Germany makes one feel, with our President, that the technique should be revised in some way so as to bring it into line with that of the Germans'. It is again interesting to note the number of patients with a positive reaction yet with no external stigmata pointing to inherited syphilis. Recently I saw a case of a father who had had syphilis. He had had two children, one of whom died of epilepsy and the other is a very well-marked mental defective, yet without external signs of inherited disease. The paper is suggestive and gives evidence of good work in a field much neglected in England.

The PRESIDENT: I congratulate Dr. Stephens upon his paper, which is a valuable contribution to knowledge at the present time. I wish that it had appeared before the Report of the Royal Commission had been published. The high percentage obtained by Dr. Stephens may be due to several causes. It is necessary first of all to separate cause from coincidence; a positive Wassermann reaction of the blood, the cerebrospinal fluid not having been examined, does not necessarily prove that the idiocy was due to syphilis. In support of this statement, I may mention that Sir John Collie has shown that 9.2 per cent. of males apparently healthy applying for employment in the various occupations connected with the administration of the L.C.C.—for example, tramway, fire brigade, &c.—gave a positive Wassermann reaction. The sera of these men were tested in the pathological laboratory of the L.C.C. under my direction, and the original technique (Wassermann's) was employed. I have tested the bloods from 200 cases of mental defectives, and have found a positive reaction in 12 per cent. I wish that Dr. Stephens had employed the original Wassermann technique, for it was laid down by the Royal Commission, strengthened by a report of a Committee of the Pathological Section of this Society, that short cuts were undesirable, and that it would be better if the original technique were employed in all investigations. I would accept the 12 per cent. of marked positive reactions of Dr. Stephens, for that about corresponded with the results obtained by Dean and others. I think the

remainder of the cases which gave a partial or a weak reaction should be reinvestigated by the (approved) method of technique I have alluded to. I presume he considers that these positive cases were due to congenital syphilis, and not acquired. I am therefore surprised to learn that the larger number of positive cases occurred in adolescent patients. Plaut's observations on the children of general paralytics rather showed that the reaction tended to disappear with age. Dr. Stephens remarked that few cases showed any signs of syphilis on the body. Dr. Plaut only found one case out of twenty infants with a well-marked positive reaction showing any eruption on the body. Infantilism, imperfect development, or arrest of development of the reproductive organs is very common in idiots and low-grade imbeciles; this may be due to syphilitic infection of the organ. I have found the spirochaetes in the testes of a fetus. The family history showed a number of pregnancies resulting in defective children, and children born dead or dying in early infancy. The mother, however, was an imbecile; the dead children and abortions came after the living, so that probably the mental deficiency had nothing to do with congenital syphilis. Indeed, the history tended to show that the mother was syphilized after having given birth to living children, including the mental defective. The serological test in such a case would have been most valuable. I should like to ask Dr. Stephens whether he has made a comparative analysis of the family histories of the positive and negative cases, especially in regard to the result of pregnancies in the mothers. When I visited Darenth I found idiots of whom it was said there were no signs of syphilis, yet examination of the fundi showed choroido-retinitis. The reason why more mental defectives do not occur as a result of congenital syphilis is due to the fact that if the spirochaetes enter the central nervous system the child dies.

Dr. STEPHENS (in reply): The paper I have written, as you see, is a bulky one. I have therefore only chosen those sections of it that I thought would be easier to read, and questions concerning others I hoped to answer as they were raised in this discussion. I have also purposely omitted all reference to a few important aspects of the subject, hoping thereby to make certain of being asked about them. Dr. Percy Smith has raised an important issue, and in answer I should like to say that the patients have been very carefully examined from the clinical standpoint, and that in *no case* could I find any definite syphilitic stigmata, lesions usually considered to be characteristic of the inherited disease. I must, however, state—and this will answer the President's question also—that the ophthalmoscopic and otological examinations of these patients were unfortunately not undertaken because of the obvious practical difficulties of such inquiries in aments precluding one from making any general statements on these points. The family histories have also been carefully investigated and were "negative," from the information I had at my disposal; but such information is invariably deficient, and in the majority of cases unreliable. These observations, based on the clinical aspects and family histories, agree with those obtained by other workers, who may be divided into two groups—those who recorded their observations before the complutic reaction was used

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in the investigation of amentia, and those who have themselves employed the test in such investigations. Dr. Shuttleworth has given us his own results published in collaboration with Dr. Fletcher Beach in 1892, when clear evidence of inherited syphilis was found in only 1.17 per cent. of their cases; while Dr. Langdon-Down detected the stigmata in 2 per cent. of cases; and Dr. Ireland recorded with some surprise that the disease was not concerned in the ætiology of amentia. Later, however, Dr. Sherlock obtained satisfactory evidence of syphilis in one or other parent in 14.4 per cent. of ninety cases, and Professor Th. Ziehen, of Berlin, detected the disease in as many as 17 per cent. of aments. Therefore, before the advent of the compluetic reaction the percentage of syphilitic cases, based on clinical evidence alone, was considered to be small. Since the employment of the test, however, a larger number of syphilitic cases has been detected, but the same small percentage of clinical results has been obtained, as shown in the writings of Dr. Plaut, Major Mott, and others. Therefore one of two conclusions is true—either that the reaction is not associated with syphilis, which in the light of our present knowledge would be considered absurd, or that the more obvious clinical stigmata of syphilis are not found in aments, which, with the testimony of the present and past experience of competent observers to support it, would appear to be correct. With Dr. Percy Smith's remarks on the causation of mongolianism most observers would to-day agree, but it was not so very long ago when this condition was considered to be parasyphilitic, and that is why I have emphasized the point that in the sera of over 72 per cent. of mongols I have found the reaction negative. In answer to the President I have to say that with the exception of seven, the patients came from all parts of England—from villages, towns, and cities; and they were drawn from all ranks of society. These points I have mentioned in my paper. Both Major Mott and Dr. Shuttleworth have referred to the technique employed in these tests. One of the principles of the "original method," as defined by the Society's Sub-Committee, is that the ingredients of the test should be derived "from different sources." If this means "from different individuals," then the technique I employed agrees with that of the "original" test. But if it is intended to mean "from individuals of different species" (e.g., the rabbit, the sheep, the guinea-pig), then the technique I employed does not agree with that of the "original" test, for I used in my "hæmolytic system" human red blood corpuscles (always obtained, it must be remembered, independently of the bloods to be examined). Otherwise my technique conformed with the principles of the "original" test, and as shown in my paper everything was done to render the determinations as accurate as possible. Major Mott questions the accuracy of the "unheated" reactions. I agree with him, but I do not think they in any way invalidate the final interpretation of the results obtained. I should like to emphasize the fact that 12 and not 42 should be taken as the total percentage of the positive results. I have recorded the other reactions merely to indicate that they have been obtained, and in my paper I have given reasons why they should be rejected as "negative."

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(February 2, 1916.)

The Use of Condensers in the Diagnosis, Prognosis, and Treatment of Nerve Lesions.

By FRANCIS HERNAMAN-JOHNSON, Captain (Temp.) R.A.M.C.

THE terms "faradic response," "galvanic response," "normal reaction," and "reaction of degeneration" are, of course, familiar to all. For half a century electricity has been used as an aid to the diagnosis and prognosis of nerve lesions. Very skilled and experienced men have been able to claim a fair degree of success, but failures have been numerous, so much so that not a few eminent surgeons and neurologists have stated that the method has little or no value. For this latter view two facts are chiefly responsible—the utter crudity of the apparatus until recently in use, and the tendency to look upon electrodiagnosis as a thing apart, and not as something to be fitted into its proper place in an ordered scheme of investigation.

A current of electricity flowing through a voluntary muscle produces no visible effect so long as it remains steady. When the potential is suddenly altered a "twitch" or single contraction occurs. At voltages of 25 and upwards the normal muscles will respond to impulses which fall to zero in a very brief period—some thousandths of a second. Other things being equal, the intensity of the contraction in a normal muscle is most affected by the voltage. But in the case of a muscle which has been for some time cut off from its motor cells in the spinal cord, the time factor assumes the chief importance. Voltage continues to exercise an influence, but however much it be raised the muscle will not contract unless the electric impulse lasts a certain minimal time.

A faradic coil may be described as an instrument by which an evenly flowing current of low voltage is transformed into a series of short discharges of relatively high potential. The voltage of the interrupted current, as led off from the secondary terminals, is determined by the intensity of the current circulating in the primary of the coil and by the construction of the coil itself as regards number of turns of wire and other details. The duration of each impulse and the number delivered per second are matters which depend upon the working of the interrupter. The former was assumed by the late Dr. Lewis Jones to be in the neighbourhood of $\frac{1}{800}$ sec., so far as its effective portion is concerned. The latter varies between 20 and 50 per second. As regards the duration of each impulse, however, the above estimate can only be taken as approximate, and the time may in some cases be much longer.

It is often said that a given muscle "responds feebly to faradism." No particular type of coil is specified, nor is any statement made as to voltage in the primary. And even if these data were given no one can be certain, with the platinum hammers commonly in use, what was the duration of the impulses used, nor—a point not without importance—the *rate* at which they were discharged. The phenomenon described—"feebleness of reaction"—is not one about which different observers are likely to be in exact agreement. In practice, of course, judgment is helped by a comparison with the sound side, if there is one, or with one's own muscles. The hopelessly unscientific nature of the whole proceeding may perhaps be best appreciated by analogy with another well-known means of diagnosis. Conceive men using clinical thermometers without scales, filled sometimes with mercury and sometimes with spirit, and having varying bores of unknown dimensions. Individual physicians might, and no doubt would, become remarkably expert in the use of the particular instrument which they possessed; but records of their cases would be of only small value to other observers. The faradic coil is doomed as an instrument for diagnosis, although its rapid succession of impulses and relatively high voltage must not be lost sight of in designing a successor. The galvanic battery or its equivalent will probably survive, as the continuous current lends itself to exact measurement. The customary record of milliamperage alone, without taking into account voltage or resistance, is, however, almost worthless; to this subject I shall return later when discussing condenser discharges. Even if accurately controlled, the continuous current cannot enable us to explore the whole diagnostic field which lies

beyond the range of ordinary faradism, for in it are many fine shades of abnormality which call for great range and accuracy in the instruments used to detect them.

The object of the foregoing remarks has been to show that, up to very recently, the first essential to the placing of electro-diagnosis on a sound footing has been absent. *The use of precise instruments in an agreed manner to secure definite and readily recognizable results is a condition of scientific progress.* The experience of one man, however learned, is never sufficient to establish a new truth unless the phenomena on which he bases his deductions can be reproduced and confirmed by his confrères. The advent of the condenser system of nerve-testing enables us for the first time to fulfil approximately these requirements.

Let it be said at once that this system introduces no entirely new principle. A condenser may be regarded as a means of storing a definite quantity of continuous current, as one might store water in an elevated reservoir. If we imagine a series of reservoirs of increasing size, containing fluid at the same level, and all having outlet pipes of the same bore, it will be obvious that the time taken by each to empty—to discharge itself—will be determined solely by its size or "capacity." In the case of condensers the material of the discharging circuit takes the place of the pipes, and the resistance of this material is the equivalent of their bore. That is to say, so long as the conditions in the discharging circuit do not vary, the length of time taken by various condensers to empty themselves is proportional to their size. When the resistance and the discharging circuit remain constant the amount of energy flowing in that circuit at any given instant is always the same, no matter what the size of the condenser may be, but the larger the condenser the longer the flow will last. If the resistance is diminished the flow of current is greater, but lasts for a proportionately shorter time. A condenser, A, twice the size of another condenser, B, may give double the amount of current for the same length of time, or give the same amount of current for double the time. It cannot, under any circumstances, give an impulse lasting double the length of time and simultaneously an increased quantity, for such a thing would in effect be creation of energy.

The method of condenser nerve-testing worked out by the late Dr. Lewis Jones is based primarily on time values. He chose a fixed or standard potential—100 volts—and assumed that the body resistance was always in the neighbourhood of 1,000 ohms, and then selected twelve condenser capacities so graded that their discharges ranged in length

from $\frac{1}{24000}$ sec. to $\frac{1}{200}$ sec. (see table). By a device which I need not now detail, he secured a further three grades of $\frac{1}{100}$ sec., $\frac{1}{70}$ sec., and $\frac{1}{30}$ sec. He found that many muscles which responded well to faradism were indeed not normal, and that those irresponsive to it showed wide differences in the length of the condenser impulses which were required to excite them. This time standard has been criticized on the ground that not only were longer impulses necessary to excite injured muscles but larger condensers, and that it was impossible to say which factor was the more important. This criticism displays confusion of thought, for the larger condensers, as it were, use up their largeness solely in maintaining their flow of current for a longer period.

TABLE OF CONDENSER VALUES, &c.¹

No.	Capacity	Discharge duration
1	0.016 microfarad	$\frac{1}{24000}$ sec.
2	0.025 "	$\frac{1}{18000}$ "
3	0.05 "	$\frac{1}{8000}$ "
4	0.062 "	$\frac{1}{6000}$ "
5	0.08 "	$\frac{1}{4800}$ "
6	0.125 "	$\frac{1}{3600}$ "
7	0.25 "	$\frac{1}{2400}$ "
8	0.33 "	$\frac{1}{1800}$ "
9	0.5 "	$\frac{1}{1200}$ "
10	0.66 "	$\frac{1}{900}$ "
11	1.0 "	$\frac{1}{600}$ "
12	2.0 "	$\frac{1}{300}$ "

The system of reckoning degrees of injury in muscles by determining the length of condenser impulse necessary to excite them is free from fallacy only so long as the voltage and resistance remain constant. The former factor is easily controlled; the latter is subject to some variation. Lewis Jones's assumption of a body resistance of 1,000 ohms holding good approximately for all condenser impulses has not been confirmed by experience. In practice, however, it is nearly always possible to achieve a minimum resistance, which does not differ greatly between one person and another, by first soaking the limb in hot water (or applying hot fomentations) and then rapidly running up to high condenser numbers. After several long impulses have been thus discharged a rest of one minute is given to the muscle. One then works backward until the shortest impulse is arrived at which will cause an ascertainable

¹ Reproduced from Lewis Jones's original paper, *Proc. Roy. Soc. Med.*, 1913, vi (Electro-Therap. Sect.), pp. 49-59.

contraction. At the Cambridge Hospital, the reaction of the muscle is recorded in a kind of shorthand—thus: "Tibialis anticus, No. 10, L.J.S., 100." The initials indicate "Lewis Jones scale," and the number refers to the particular stud on the Lewis Jones instrument. These numbers have been attacked as "meaningless." On the contrary, their meaning is most precise. Written at full length, No. 10 means that, under certain standard conditions, subsequently to be detailed, the particular muscle investigated requires the impulse from a condenser having a capacity of 0.66 microfarad, charged at a potential of 100 volts. The universal use of the numbers 1 to 15, so far from being confusing and unscientific, on the other hand, would compel that very standardization of instrument and uniformity of method which have been hitherto so conspicuously lacking, which it is the chief object of this paper to advocate.

The original Lewis Jones instrument for diagnosis charged and discharged its condensers by means of a metronome. The muscular contractions which it was possible to produce were therefore limited to a series of single twitches similar to those produced by making or breaking a continuous current. Now it is well known that in the old system of nerve-testing the response to coil currents sometimes returns, while that to battery currents still remains feeble. In coil current we have the effect of summation of stimuli, due to the rapid discharge into the muscle of a succession of impulses at a high voltage. In the case of a coil none of the factors concerned can be measured. In the modification of the Lewis Jones instrument which I have introduced, any number of impulses can be produced between 1 and 20 per second or more, 20 being the minimum number to produce tetanization. This number is recorded, the nature of the individual impulse is under control, and the voltage is known. Thus an exact record can be made of the condition of a test. I have not yet seen a muscle which would respond to faradism which would not respond to tetanizing condenser discharges at 100 volts. If such should occur, however, I have little doubt but that it would respond, at some potential between that and 200.

In order to obtain discharges of a longer duration than $\frac{1}{200}$ sec., Lewis Jones used his last three studs over again at a voltage of 200, and through a resistance of 5,000 ohms. The modified instrument which I have mentioned was constructed some months ago, and is arranged to make this transfer automatically, three extra studs being added for the purpose. Although this is convenient, it is accomplished at a sacrifice of flexibility, and I propose having it altered so that any

voltage may be used on any stud, and also a definite resistance thrown in wherever desired.¹

This may seem at first sight a contradiction of what I have said with regard to standardization, but it must be remembered that we are not yet certain about our standards in all cases. My own experience is practically confined to the upper and lower extremities. In the arm a standard voltage of 100 gives excellent results in diagnosis and prognosis. In the lower limb the same voltage has, on the whole, been satisfactory, but anomalies have been encountered. Major Bailey, of Brighton, informs me that he has had several cases involving the facial muscles and the tongue, and that he has had to adopt a lower charging potential in order to obtain good results. The converse appears to be sometimes the case in the lower limb, and we may perhaps have to adopt a standard voltage of 125 or 150 in testing thigh and leg muscles. Meanwhile the original 100 volts may be regarded as entirely satisfactory in the upper extremity.

So far I have dealt with the *standardization of the instruments* used in electro-diagnosis. That they should be employed in a *similar manner* if the results obtained by one observer are to be compared with those of another is a matter of equal importance. Complete uniformity is perhaps too much to hope for, but at least any method adopted should be simple enough to permit of its being accurately carried out from a written description only. The following procedure is used at the Cambridge Hospital:—

(1) *The Electrodes.*—Rectangular pads are used, 2 in. by 1 in., which are moistened with tap-water. Both electrodes are placed on the muscle under examination, one on the motor point and one elsewhere. When possible they are pressed flat on the skin; but in some cases, as with the extensors of the thumb, when these are very feeble, it is necessary to pinch the muscles up between the electrodes in order that their faint contraction may not be obscured by spreading of the necessarily powerful impulse used to excite them.

(2) *Preparation of the Patient.*—The application of moist heat has already been mentioned as helping to reduce skin resistance. It is also often necessary for another reason—namely, that the excitability of a muscle is much reduced by cold. The patient himself should be fresh, so that the morning is usually the best time for an investigation.

¹ The modified Lewis Jones instrument, fully equipped with all the devices found useful by the author, has been placed on the market by Messrs. W. Watson and Sons, 196, Great Portland Street, W.

If a muscle is obviously over-stretched, as by uncorrected foot-drop or wrist-drop, it should be given a few days' rest in a "relaxation splint" before a test is made. When such relaxation is not possible owing to adhesions or contractions, the prognosis is often less unfavourable than might appear from the nature of the condenser reactions.

It will be noted that the attempt to formulate a standardized electrical procedure presents one point of theoretical weakness—the factor of indeterminate resistance. Nevertheless, in practice this does not



Hernaman-Johnson's modification of the Lewis Jones condenser set, with voltmeter and rheostat to charge condensers at any voltage between 25 and 200. Also speed regulator to give any number of revolutions between 1 and 30 per second, and speed recorder. The patient's circuit is earth-free.

seem seriously to affect the comparability of results, the reason being that the utmost care is taken always to give any suspected muscle the best possible chance. To borrow a legal analogy, we assume that it is innocent until the most careful investigation has proved it guilty.

An account of the uses of condensers in nerve- and muscle-testing would not be complete without a reference to the use of the continuous

current. For our present purpose a battery yielding a constant current at 100 volts may be conceived of as adding to our series of condensers one of infinite capacity and consequently of infinite duration of impulse. But an impulse lasting only $\frac{1}{200}$ sec. at 100 volts is already somewhat painful; so that we cannot, in fact, use such a voltage for continuous current testing, but must at least cut it in half. A muscle contracts when a galvanic current is "broken," but the factors concerned elude definite measurement. The conditions under which the excitation at "make" is produced are, however, capable of being accurately recorded. As the test is at present carried out only the milliamperage is stated, with perhaps the size of the pad. But it is also necessary to know the voltage, as the electric pressure required to drive a given milliamperage through the skin will vary a good deal, according to the resistance of the latter. If we wish to reproduce the conditions of a given test, we must devise a means of controlling the variable factor—i.e., the body resistance. This can be done by choosing a standard, say 2,000 ohms, and always bringing the total resistance of the current up to this figure by means of an adjustable rheostat. Perhaps in future it may be found possible to apply some analogous arrangement to standardize the resistance in the case of condensers. The use of the continuous current in a scientifically accurate manner offers a field for further research.

In the absence of a professed neurologist at Aldershot, I was called upon very early in the War to give what assistance I could in the diagnosis and treatment of nerve injuries. The Lewis Jones apparatus, although then obtainable only in a somewhat crude form, appealed to me as likely to afford more exact information than coil and battery testing, and I requisitioned one for this electrical department. At that time there existed practically no data as to the meaning of condenser reactions in nerve injury, some observations in connexion with infantile paralysis having alone been published. In seeking to build up a system by which condenser reactions might be made of practical help in military work it was, of course, necessary to have some sort of working hypothesis. The one chosen was to this effect: that when a muscle needed very long condenser discharges to excite it the motor nerve was cut, or so injured or involved in fibrous tissue as to be completely blocked; and that when medium discharges were required a minor degree of trauma had occurred. It was further assumed that when the condenser reactions improved month by month, recovery was taking place, even in the continued absence of voluntary movement; and that when these reactions did not so improve, the prognosis, apart from

operation, was bad. These assumptions had proved very useful, and, subject, it is true, to numerous exceptions and qualifications, have in the main been borne out by seventeen months' experience.

I need hardly say that it is not possible to distinguish between anatomical and physiological block. A much more important practical limitation is this. It is not possible to say whether a block actually exists at the time of the test. All that can be definitely stated is that, when a muscle responds only to No. 11 and upwards—i.e., to impulses lasting $\frac{1}{100}$ sec. or longer—there has been at some time a complete blockage of all motor impulses for some considerable period, probably weeks.

It would appear that when a motor nerve is "blocked" for any length of time, the muscles exclusively supplied by it undergo certain changes which prevent their response to the very short electrical stimuli which normally excite them. If the change in the electrical excitability always disappeared coincidently with the re-establishment of a path for motor impulses, it would be highly convenient for the diagnostician. But Nature has not arranged it so. It is quite possible for the condenser reactions to remain stationary during the healing of a nerve, or even to improve somewhat, and then actually retrogress with the return of voluntary power. This phenomenon is most often seen after the freeing or suturing of a nerve. I have only once seen it occur in a case that was healing spontaneously. There is but one electrical phenomenon which I think may prove to be pathognomonic of complete blockage existing at the time of the test. When the condenser number required is 12 or more, and when the contraction of the muscle falls to zero after four or five progressively feebler efforts, operation has so far shown complete severance of the nerve trunk. The non-occurrence of this phenomenon is, however, no evidence against severe injury.

The fact that high condenser numbers may be required to excite a muscle the motor nerve of which is far advanced towards recovery might at first sight appear seriously to invalidate the diagnostic value of such reactions. In practice this is not the case, provided that the condenser response is regarded as an important, but not exclusive, means of determining the broad issue as to whether or not operative interference is desirable. The test is unreliable at any less period than six weeks after the injury. Faradic response usually vanishes in a week or ten days, but a muscle severed from its motor nerve will respond to 7 or 8, condenser scale, just after it has lost its response to coil currents. Reactions between 7 and 9 occurring in a case two or three

months old would be regarded as evidence in favour of possible spontaneous recovery; but if found at the end of a fortnight or so are just as likely as not a stage in the downward path towards 12 and beyond. A case of gunshot injury near the elbow which I saw at the Cambridge Hospital is interesting as illustrating this point: The patient was sent to the electrical department with a letter saying that his wound had occurred three months before. There were no sensory changes. The extension of the forearm responded to No. 9 condenser scale. A report was given to the effect that the musculo-spiral was certainly not divided, but might be involved in fibrous tissue. Operation showed a completely severed nerve. It was then discovered that the wound had been received only three *weeks*, not three *months* previously. A test made one month afterwards showed no response below 12.

Most patients, however, for various reasons are not sent to the electrical department until their injury is two or three months old. Cases showing reaction of 12 or upwards are carefully examined for any trace of voluntary movement. An attempt is also made to stimulate the nerve itself above the lesion when this is anatomically feasible. If there is no movement, no nerve conduction, and also complete interruption of sensory impulses operation is obviously to be recommended, provided the surgical conditions permit of it. Often, however, the sensory path is still, to some extent, intact, and there is perhaps even faint conduction in the nerve. At first we used to give such cases a chance of recovery, carefully observing and treating them for some months. Nearly all have had eventually to be operated on and have shown definite involvement of the nerve in scar tissue. Our practice is now to operate unless delay is advisable for other reasons, such as the state of the wound.

When the reactions are between 8 and 10, inclusive, a varying period of waiting is prescribed, but cases which show no detectable improvement of any kind in two months should, as a rule, be operated on.

In a period of twelve months twenty-five cases were recommended for operation. In twenty-two of these definite injury of the nerve was found, chiefly in the form of involvement in fibrous tissue. In two the nerve looked healthy, but the bullet had passed close to it. We know that in the case of muscles they may appear sound and yet be seriously damaged within, so that it is reasonable to assume that hæmorrhages may occur within a nerve without altering the appearance of the sheath. In the remaining case in which nothing was found the bullet had passed at some distance from the nerve trunk.

The last patient referred to had been wounded in the thigh; in another somewhat similar case the patient refused operation, and after some months began to show signs of recovery, though whether this will be sufficient without surgical interference is still doubtful. No similar cases have occurred in the upper extremities. This makes me wonder if the standard of 100 volts is sufficiently high for use in the lower limbs; only further experiment and observation can determine this point.

In estimating the chances of recovery without operation in any given case I endeavour to the best of my ability to take all factors into consideration; but the fact remains that my opinion is based very largely on the condenser reactions, interpreted in the light of what has been found at the operation, and in the course of cases in which for some reason an operation, though recommended, has not been performed.

It is worth noting that musculo-spiral injuries have been very common in this War, that in many of them electro-diagnosis is the only method of investigation open, and that as a class they responded remarkably well to operative treatment—signs of voluntary power sometimes returning within five months after secondary suture, and in a few weeks after freeing from fibrous adhesions.

The electro-therapist should attend operations on nerves whenever possible, not only for the purpose of seeing for himself what is found, but in order to gain further information by direct stimulation of the exposed trunks. This procedure might well in itself form the subject of a paper. I mention it only to call attention to its importance, and to point out that the standardization of the currents used is not of less importance here than elsewhere.

I will now deal very briefly with the treatment of nerve injuries in so far as the condenser apparatus is applicable. In its original form only a succession of single twitches could be produced in a muscle, such as are obtained by making and breaking a continuous current. The modified apparatus to which I have referred can, if necessary, produce tetanizing current, and this current causes extraordinarily little skin feeling relatively to the amount of muscular excitation, even when quite small pads are used. As the individual impulses may be of any length up to $\frac{1}{30}$ sec., the tetanization of practically any muscle is possible, even though it be completely cut off from its motor nerves. Such a power must be used with discretion, but when electrical stimulation is desirable at all rhythmical tetanization is infinitely superior to treatment by what are popularly known as galvanic twitches.

The careful picking out of the individual muscle is the key to success in a large number of cases, and is often neglected because it requires time and skill. Such work can rarely be left in the hands of a lay assistant. It is so easy on the one hand to produce over-fatigue; or, on the other, to fail in provoking all the required movements. I have watched work of this kind being done. I have seen the commission of almost every kind of therapeutic sin—including stimulation applied ostensibly for the benefit of weakened extensors, which succeeded only in producing vigorous contraction of their normal opponents by spreading of the strong currents employed. If the stream of impulses from the condenser is more powerful for good than the older methods, it is also, wrongly used, capable of doing proportionately greater injury.

Returning once again to the question of diagnosis and prognosis, I should like to make it quite clear that I make use of sensory investigations so far as my knowledge of them goes. There has, I think, been a tendency of late to give electrical reactions a very secondary place. Some of the reasons for this distrust I have tried to point out, and at the same time to show that most failures are due to defective instruments and faulty methods. That an expert neurologist may get along quite well with only a very moderate amount of electrical knowledge may be conceded. But the converse is also true, as I have proved, at any rate, to my own satisfaction and that of the surgical staff of the Cambridge Hospital. The perfect physician would, no doubt, combine with an expert knowledge of the nervous system a complete familiarity with electrical principles and technique. But such prodigies are rare, and one is certainly justified in asking that nobody should speak lightly of modern electro-diagnosis unless he has thoroughly familiarized himself at first hand with the technique employed, and the results obtained by someone who has had extensive experience of the method and who believes in its value.

In bringing this paper to its close, I venture to submit for consideration the following conclusions:—

- (1) Electrical reactions are, and must remain, important in diagnosis; they cannot conceivably be dispensed with.
- (2) The older methods need drastic revision in regard to instruments, technique, and terminology.
- (3) The Lewis Jones condenser instrument, as modified to give either slow or rapid impulses, is an accurate and scientifically standardized substitute for the faradic coil, and also, in the great majority of cases, for the galvanic current.

(4) Regarding nomenclature, the terms "faradic response" and "reaction of degeneration" should be dropped and their places taken by a simple statement as to the smallest condenser which will produce any appreciable contraction under given conditions.

(5) As to the standard to be adopted, the condenser values originally observed by Lewis Jones appear to do very well, and should be adhered to. His voltage of 100 works excellently in the upper extremities; it may need to be a little increased as regards the leg and decreased for the face. The rule that a muscle should always be "coaxed" to do its best—i.e., to respond to the shortest possible impulse—must be carefully observed. The adoption of the Lewis Jones values would enable muscle reactions to be designated by numbers, and thus greatly simplify reports.

(6) The condenser system opens no royal road to diagnosis and prognosis. It requires careful technique, and must be intelligently applied in the light of general experience and a review of all the circumstances of the individual case. However perfect the method, much will always depend upon "the man behind the gun."

DISCUSSION.

Major FRED BAILEY (Brighton) : The "condenser" method is but one of the various devices for measuring the deviation of the condition of a muscle from its normal, but by it we believe one can obtain a more correct estimation of the character and amount of that deviation. Before its introduction we were limited to the use of the constant and the faradic currents as testing agents, and these for various reasons (the principal of which were that the impulses derived from the constant were much too long, and those from the faradic not short enough) did not give us sufficiently precise information. They only enabled us to say that a given muscle was normal, or the subject of partial or of total "reaction of degeneration"—three very indefinite and erratic groups. Now the condensers give us *twelve* groups (or more), and, according to the group we place it in, we can definitely state, by a numerical method, that any given muscle deviates to a given degree (which may be represented by a numerical fraction) from the normal. Thus, if we term a normal muscle N, these abnormal muscles may be represented as $\frac{N}{2}$, $\frac{N}{3}$, up to $\frac{N}{20}$, just as an ophthalmic surgeon reckons vision as $\frac{6}{6}$, $\frac{6}{12}$, up to $\frac{6}{20}$, and so on. The principle is a simple one : we have merely to discover, by moving certain switches backwards and forwards, the smallest electric discharge that will just cause a muscle to undergo visible contraction, and note it down. For quite accurate working (which, however, is not always absolutely essential) the required amount of electricity, producing a lowest initial visible contraction of the muscle, should be obtained (by so arranging the charging voltage and the discharging resistance) from the *lowest (or first) stud* in the condenser box. We first test the *normal* muscle on the sound side corresponding to the defective muscle on the other; next, with the conditions of charging voltage and resistance unaltered, we test the affected muscle by moving forwards and backwards the *condenser* studs only, thus increasing or decreasing the capacities of the condensers used until we find the condenser capacity on which the *damaged* muscle first visibly contracts. Then, on the sound side, the tibialis anticus, flexor sublimis digitorum or interossei, as the case may be, are reckoned as N (i.e., contracting on the lowest stud); the abnormal muscles on the affected side may be compared with it, as $\frac{N}{8}$ (if on sixth stud) or $\frac{N}{10}$ (eighth stud), which may be considered equivalent to degrees of *partial* reaction of degeneration; or as $\frac{N}{10}$, $\frac{N}{11}$, $\frac{N}{12}$, (tenth, eleventh, or twelfth stud), equal to increasing degrees of deviation from normal, corresponding to varieties of *total* reaction of degeneration. This is a correct and scientific method, and signifies a simple and easily understandable fact—namely, that if a *normal* tibialis anticus's initial contraction is produced by a particular type of discharge lasting for a certain measured period of time (the exact period does not matter), which we may term N, then

the same type of discharge in all respects, *except the length of time it lasts*—which, for example, we will say is twenty times as long—causes the initial visible contraction in the abnormal muscle, which would be consequently written down as $\frac{N}{20}$. To obtain the *normal* initial contraction on the first condenser stop requires a *variable* voltage for condenser charging and a *variable* resistance for discharging, because normal muscles may vary greatly in their irritability, and the skin resistance is also very variable. My apparatus has a charging voltage which rises, by 25 volts, from 0 to 225 volts, and a resistance (which may be added to the skin resistance), rising by 1,000 ohms at a time, from 0 to 10,000 ohms. With this apparatus the charging voltage and the resistance to the discharge can be altered quite rapidly until the normal standard, N, is reached with the normal muscle. (It was made partly by Leslie Miller and partly by Magnus Volk and Alderton, of Portland Street, Brighton, under my instructions.) It is Lewis Jones's original condenser apparatus modified.

On January 17, 1913, Dr. Lewis Jones read before the Electro-Therapeutical Section of this Society a paper on the use of condenser discharges in muscle-testing, and exhibited a condenser apparatus for estimating "reaction of degeneration" in muscles; he claimed greater accuracy for the condenser tests, as compared with former methods, and gave a general explanation of the method of working. He used a constant charging potential of 100 volts to charge his condensers, the resistance to the discharge being only that of the body, estimated at about 1,000 ohms. His box possessed twelve gradually increasing condenser capacities; the smallest one, with his constant voltage and the normal body resistance, would give an initial visible contraction with an ordinary healthy muscle. The muscles he had been testing would be ordinary muscles such as the flexor sublimis digitorum in the arm or the tibialis anticus in the leg; also for these ordinary muscles he found that (with his twelve capacities), if a particular muscle would only initially visibly contract on the highest capacity, it was in the condition generally known as "total reaction of degeneration." Now there arise two arguable questions in reference to Dr. Lewis Jones's deductions: the first is whether all voluntary muscles in the body do react in the same way as the muscles he selected; the second is whether the skin resistance is a constant. Dr. Lewis Jones found in practice that even ordinary muscles under certain conditions of extreme degeneration would *not* contract even to his highest condenser capacity. So, to overcome this difficulty, he very much increased the resistance through which the condenser discharged by adding a resistance of 5,000 ohms, and in order to make up for the loss in strength which this entailed, he doubled his charging voltage to 200 volts. Under these changed conditions he found that some of these very seriously damaged muscles could be made to contract to the three lowest capacities of his condenser, but some did not. Since then various difficulties have arisen, and more particularly it has been discovered that certain muscles,

such as the foot muscles, with his original charging voltage and original body resistance, do not contract to the lowest capacity of the condensers; consequently one has to start at a different capacity as representing the normal in these muscles. Also certain muscles, particularly of the face and tongue, are so highly sensitive that they always contract to the lowest capacity, as usually charged and discharged, and they would in all probability contract to capacities much smaller. To overcome these difficulties I had the charging-box I now exhibit constructed, which by a shunt arrangement gives charging voltages increasing by 25 volts to 225 volts, and the discharging resistance box, by which resistances of 1,000 ohms can be successively introduced, so that a final resistance (when added to the 1,000 ohms body resistance) of 11,000 ohms can be obtained. These boxes, with the original box of condensers and the metronome interrupter, complete the apparatus necessary for testing abnormality in muscle. Taking any muscle, say an abnormal tibialis anticus, the first thing is to test its normal fellow on the opposite side and to obtain an initial visible contraction from the lowest or lowest but one condenser capacity with a certain voltage and resistance—i.e., such a contraction that, by decreasing the voltage by 25 volts, the muscle ceases to give a result. We have now a normal starting point for the tibialis anticus of the individual; if the perfectly healthy muscle gives its contraction under these conditions, then the opposite damaged muscle (if it were in a condition equivalent to total reaction of degeneration) would only contract on a very high condenser capacity. Again, in a patient suffering from facial paralysis, should an attempt be made to test the facial muscles by the original Lewis Jones's method—that is, with 100 volts charging voltage—it is found that, at an early stud of the condensers, the discharge is so painful that the examination cannot be finished. Should, however, the condensers be used with a charging voltage of 50 volts only, with, if necessary, an increased resistance inserted, then on the unparalysed side of the face the normal for the healthy muscles can be readily obtained on the lowest condenser, and it is quite easy to test the facial muscles on the damaged side and obtain the correct comparison with the normal.

As another example of its use I would refer to a case brought to me from the Indian Hospital in which a piece of shrapnel was believed to have severed or severely damaged the right hypoglossal nerve. The patient had various interesting symptoms. He had sensory paralysis of the right palate and pharynx with paralysis of the right vocal cord, unequal pupils, the larger being on the left, and unequal palpebral fissures. There was great wasting of the right side of the tongue. The question put for decision was the presence or not of the reaction of degeneration of the right tongue muscles. Now, the tongue is very sensitive to pain, and as there is on the tongue practically no skin resistance, it would be quite impossible to test with the ordinary condensers at 100 volts. But, by using the condensers with only 25 volts charging voltage, and interposing a resistance of 10,000 ohms, I obtained a condenser discharge

with which the muscles on the *left (normal)* side of the tongue gave the initial visible contraction on the *lowest but one* of the condensers. This was the *normal reaction* of the patient's tongue muscles. Applying this test to the right, or paralysed side, it was found that the initial visible contraction did not occur until No. 12 stud, the largest condenser capacity in the box, was reached. The reaction was labelled $\frac{N}{130}$ and explained as equal to an advanced total reaction of degeneration.

Dr. Lewis Jones originally stated that the condenser method allowed a ready recognition of *twelve different* degrees of muscular excitability (instead of the three grades—normal, partial reaction of degeneration, and total reaction of degeneration), and under his conditions as to changing voltage and discharging resistance that the discharge duration of these condenser capacities varied from $\frac{1}{1400}$ sec. up to $\frac{1}{50}$ sec. It is evident that the variations of the length of the wave, if the wave-length of the lowest condenser be taken as the unit, are from 1 up to 120 (1, $1\frac{1}{4}$, 3, 4, 5, 8, 15, 20, 30, 40, 60 and 120), so that the method of estimating by a fraction of N is a permissible one. The point which I wish to impress is that for very accurate diagnosis the first thing is to find the discharge wave-length which will cause the initial visible contraction in the corresponding healthy muscle, preferably of the patient, or (not so good, of course) of another individual. Taking that as N, if the unsound is labelled, for example, $\frac{N}{130}$, we infer that the damaged muscle has depreciated to an extent which makes it only $\frac{1}{130}$ th as irritable as a normal muscle, and from this we can form an opinion as to the damage the nerve supplying the muscle has sustained.

Condenser testing is only *one* of the helps to diagnosis of nerve injuries; the estimation of sensory abnormalities is extremely important, of course, if the nerve is in part sensory. But sensory tests may absolutely fail in cases of malingering or the like, or in cases where it is impossible to obtain the co-operation of the patient, which has happened several times at the Indian Hospital. The estimation of the loss of *epicritic sensibility* is the most valuable of the sensory tests, and yet one sees quite frequently an attempt to estimate loss of sensation by pressing with a pencil point or a finger. Sherren has pointed out how easily epicritic sensory deviations may be overlooked. Of course, only cotton-wool or a camel-hair paint-brush should be used, for *deep sensibility* may be comparatively easily elicited in quite severe nerve lesions by any pressure or movement in the slightest degree excessive, and deep sensibility is carried *not* by the ordinary nerves, such as ulnar, median, or the like, but by nerve-fibres running with the vessels, and so its presence may quite easily lead to diagnostic error.

Dr. Cumberbatch, of St. Bartholomew's Hospital, who worked with Dr. Lewis Jones from the beginning, has advanced various criticisms on Captain Hernaman-Johnson's and on Dr. Lewis Jones's conclusions as to the

accuracy of condenser work and its value, by suggesting that we must consider the question that large condenser capacities give not only discharges of *longer* wave-lengths but also *stronger* stimuli. One does not quite follow this criticism; as most observers seem to agree that the height of the peak of the wave remains always the same so long as the charging voltage and the discharging resistance do not vary, whatever the capacity of the condenser. It is *only* the length of the wave that alters. One wonders whether anomalous *results* may not be due to the fact that the skin resistance, which has been supposed to be constant, but which on the contrary is variable, has not been reduced to its lowest limit by heat, diathermy, saline soaking, &c., as Hernaman-Johnson showed to be necessary. Dr. Lewis Jones supposed that the skin resistance to small condenser currents did not vary. Observers are now agreed that this resistance is, to a certain extent, variable. The most favourable way of getting rid of the factor of variability is to add a large *constant* resistance so that the component resistance of the variable and constant resistances would exhibit a very small variability. My own experience is, that to obtain the most accurate results, it is good to start by putting in as large an extra resistance as possible, and then to increase the charging voltage until the first visible contraction of the muscle is seen on the lowermost condenser capacity. Under these conditions I do not think the discharges of the various sizes of condenser would vary in strength but would merely vary as to length. Dr. Lewis Jones's original statement was that "the *character* of the wave of discharge of a condenser is well defined; it is determined by the resistance through which the discharge takes place and by the capacity of the condenser; *its height, but not its duration* is determined by the potential at which the condenser is charged; when the charging voltage is constant and the resistance of the circuit is constant, the waves of discharge become longer as the capacity used becomes greater, and this view evidently holds good when the resistance is sufficiently great and the charging potential is large enough to produce the muscle contraction."

One of the results of greatest value, in using the condensers, is that by them we are able to test from time to time the muscles supplied by a certain nerve, as to whether their reactions show improvement or the reverse. To give an example of a case which depends somewhat on these factors: A Canadian soldier, who was a good baseball player, was watching a game of baseball and the ball trundled to his feet. He took up the ball and threw it back to the players. It is well known that the baseball pitchers can produce extraordinary effects with the ball, making it (just as in slicing and pulling at golf) describe curves *in the air*, and the necessary muscular action must be extremely violent for the moment. In this case, as his Canadian doctor told him, "his muscles were too strong for his bones," and the humerus, in the act of pitching, sustained a long fracture. He left for England the next day; on board ship the surgeon attending him discovered paralysis of the

musculo-spiral nerve; when he arrived at the Military Hospital in Brighton he was brought up to be tested. The muscles supplied by the musculo-spiral in the forearm reacted to No. 7 stud of the condenser box, that is, what was equivalent to "partial reaction of degeneration." Although the history was definite that the onset of the paralysis was quite soon after the humerus was broken, the question arose whether the nerve had been severely contused or whether it had been torn across. It was evidently a case of waiting to see whether improvement or the reverse took place. In fourteen days the lower portion of the triceps had improved nearly to normal, but the extensor muscles in the forearm remained much as before; three weeks later the lower head of the triceps was quite normal, while the extensor muscles now only responded on No. 5 stop, that is, they were getting distinctly worse. Under these circumstances advice was given that it was advisable to cut down on the nerve and find out its condition.

I have used the condensers in the *treatment* of nerve cases. I began by using a mercury interrupter, such as is used for X-ray work. This, however, was too fast, and it appeared from personal experience that rapidly repeated condenser discharges are themselves rather painful. It occurred to me that the ordinary testing metronome interrupter, causing a simple contraction followed by rest, would be quite suitable for treatment—the essence of electric treatment is to exercise a particular muscle and give ample rest between contractions. It is extremely important in all electric treatment that *healthy* muscles should *not* be caused to contract. I consider that the greater portion of electric treatment as carried out by nurses, masseurs, and others is, in all probability, much more harmful than beneficial. Any electric current discharge, even if used with the greatest care, overflows and causes contraction in opposing healthy muscles much more rapidly than in the abnormal muscles, which are the only muscles one wishes to act upon, and contraction of opposing healthy muscles is distinctly harmful to those paralysed. I think it was Major Robert Jones, of Liverpool, who pointed out that one of the most valuable factors in the treatment of these cases was the promotion of relaxation of abnormal muscles by means of a splint or other apparatus. No damaged muscle is likely to get rapidly well if the opposing undamaged muscles are continually stretching it.

Dr. E. P. CUMBERBATCH: The introduction of the method of testing electrical reactions by condenser discharges marks a real advance. Science is measurement, and in place of the stimuli provided by the faradic and galvanic current, both of which are unmeasured, we make use of the measured stimuli provided by condenser discharges. There were certain points in Captain Hernaman-Johnson's interesting communication on which I should like to speak, but as my remarks would deal with details of apparatus they would be out of place at the present meeting. I should like to protest against the use of the words

"stud 8, stud 11," and so on, when giving a report on the electrical reactions furnished by the condenser method. These numbers convey no accurate meaning. Why not refer to the capacity of the condenser that is required to evoke the response of the muscle, or the duration of the stimulus that such a condenser supplies? The fact that a muscle requires the discharge from a large capacity condenser does not, I think, necessarily indicate that its motor nerve has received severe injury, requiring surgical operation for repair. I have seen many cases of paralysis in which the muscles required the largest capacity condenser, charged to 200 volts, and with 5,000 ohms in the circuit, make spontaneous recovery. An interesting feature about these cases was the persistence of reaction of degeneration for some months after voluntary power had returned. It is therefore possible for a muscle to have the reaction of degeneration (so-called), requiring the largest capacity condenser, and yet be under voluntary control. This has been my experience of *medical* cases in which the nerve has been inflamed. I have seen it also in surgical cases in which the nerve has been injured. I have found that most cases showing the persistence of reaction of degeneration with the recovery of voluntary power show excitability of the *motor nerve* to electrical stimuli. Personally, I am of the opinion that the response of the motor nerve supplying the paralysed muscle will give more information regarding diagnosis and prognosis. We test muscles for the purpose of obtaining information on the condition of their motor nerves, why not test when possible the nerves also, above and below the injury? A muscle that has been cut off from the influence of the spinal cord grey matter by an injury to its nerve will show reaction of degeneration, and the nerve below the injury will gradually lose its excitability. The condition of the motor nerve in a case of injury can be tested at intervals, and the condensers, providing as they do *measured* stimuli, will show whether the excitability is increasing, remaining stationary, or diminishing till it becomes extinct. If the excitability is diminishing or is extinct, and remains extinct, surgical exploration would be called for, especially if other methods of examination also indicated this procedure. When a motor nerve trunk has been exposed during surgical exploration it should be tested electrically in the theatre, especially if it shows no gross injury. The functional condition of the nerve can then be examined. In the electrical treatment of paralysis, whether surgical operation has been performed or not, the nerves should receive treatment as well as the muscles. Cases of injury of the nerves should receive treatment by the constant current as soon as possible, and this will undoubtedly aid the absorption of inflammatory products and so tend to prevent the formation of scar tissue. If scar tissue has formed, chlorine ionization should be tried, and it should not be left off even if an operation has been performed for the removal of the cicatricial tissue. Such treatment cannot, of course, be applied by the condenser method. Condenser discharges can, however, be applied for the treatment of the muscles. It may be asked

whether it is necessary, when carrying out the treatment of the paralysed muscles, to make them actually contract. Personally, I do not think that it is necessary. At St. Bartholomew's Hospital most of the cases of paralysis that are treated in the electrical department receive the sinusoidal alternating current, whether the muscles show reaction of degeneration or not. The muscles with reaction of degeneration do not contract when stimulated by the current mentioned, and yet good results are obtained.

Captain HERNAMAN-JOHNSON (in reply) : With most of what Major Bailey has said I am in absolute agreement. I have already referred to his views as regards the face, and stated that a higher voltage than 100 may be found desirable as a standard in the lower limbs. I fear that I cannot express equal agreement with the remarks of Dr. Cumberbatch. I remain quite impenitent as regards the use of numerals in reports. I have already explained that they are merely a convenient shorthand to designate definite values as arranged by Dr. Lewis Jones in his original instrument. I maintain that this instrument is already sufficiently well known to justify abbreviations in referring to it. Dr. Cumberbatch has pointed out that some workers are 'experimenting with other condenser values. Such experimentation is praiseworthy among the elect, but, in the meanwhile, nerve injuries have to be dealt with literally by the thousand. It is quite obvious that many, if not most, of the men called upon to deal with them will have to learn this special work *de novo*, and to such a clear lead must be given. If we wait for the ideal system the War will be over. I speak from experience when I say that the condenser values as introduced by the late Dr. Lewis Jones have proved eminently satisfactory in practice. As to the fact that high condenser values may occasionally be necessary to cause muscles to contract which already show voluntary power, I dealt at length with this in my paper. In military hospitals we find the condenser method a vast help in determining the broad question as to whether or not cases require operative interference. The fact that, in a few cases, the operation may prove to have been unnecessary cannot be helped ; it is a small price to pay for the time saved in the great majority. The condenser method can only lead us seriously astray if all other considerations are neglected. I was at pains to mention the importance of testing a nerve during the operation. We use a tetanizing condenser current, with both electrodes on the exposed nerve. The latter is isolated from the surrounding tissues by the sterilized rubber. I agree that nerves as well as muscles should be stimulated when this is feasible. But even when some nerve conductivity remains—as it does in partial strangulation by fibrous tissue—operation may still prove necessary ; and it is here that the reactions of the muscles help. With regard to treatment, it is not for me to dispute the good results that Dr. Cumberbatch has had with the sinusoidal currents of a strength not sufficient to produce muscular contraction. I can only say that, *where it is desirable to stimulate a muscle at all*, my

experience is strongly in favour of a tetanizing current. In pre-condenser days I have cured by faradic stimulation many cases of paralysis which had failed to improve under treatment by the constant current and by galvanic twitches; and, since the War, I have found a tetanizing condenser current particularly valuable in cases where the faradic current just failed to excite, or where an intensely strong and unpleasant faradic current is required. I need hardly say that the tetanization is always *rhythmical*, and that great care is needed not to overtire a damaged muscle. I am a firm believer in the use of continuous current at the site of the nerve injury, applied so as to pass transversely through the limb. I discussed its use in an article on treatment in the *British Medical Journal* in July last; the matter was not mentioned in my paper to-night, as my remarks were concerned solely with the employment of condensers.

Surgical Section.

SUB-SECTION OF PROCTOLOGY.

President—Mr. F. SWINFORD EDWARDS, F.R.C.S.

(May 10, 1916.)

Rectal Wounds in the Present War.

By CUTHBERT WALLACE, Colonel A.M.S., F.R.C.S.¹

WOUNDS of the rectum can roughly be divided into two classes: (a) Those that may be said to complicate wounds of the buttocks, ischial fossæ, or perineum; and (b) those that are caused by missiles which traverse the pelvic portion of the abdomen. Both are often complicated by fractures of the bony pelvis.

(a) The missile may be a bullet or shell fragment. If the latter, the external damage may be very great and a large portion of the gluteal region torn away. The sphincter and lower portion of the bowel may be completely torn away, or the lumen opened on one side only. Again, with small missiles the sphincter may be intact and the lumen opened, or the tube perforated above that muscle. The peritoneum may or may not be opened. The danger mostly to be feared is from septic absorption, but on the whole the extensive wounds of the buttocks do not do badly at the Front, as they are widely open from the nature of the injury.

Treatment.—The treatment of the lesser perforating wounds follows ordinary lines, and consists in getting free drainage by laying open the track or dividing the sphincter (Makins). If the peritoneum has been opened something may be gained by cutting off the abdominal cavity

¹ Consulting Surgeon, Expeditionary Force in France.

by sutures. The question of colostomy must be considered and its expediency must be determined by the difficulty or ease of keeping the patient clean and comfortable. It will mostly be called for in those cases in which the whole lower segment of the bowel has been carried away and the torn end of the bowel lies, as it were, patent in the pelvis. In such a case the lumen can be closed by sutures or a purse-string ligature, which will, at all events, tend to prevent contamination of the pelvis while adhesions are forming. A transverse colostomy is the operation of choice for two reasons: (1) If a permanent artificial anus is necessary the median transverse opening is most convenient both from the ease of cleaning and of fitting some sort of belt; (2) if the colostomy can be closed the freedom of the transverse colon greatly facilitates the operation.

(b) Under this heading it is convenient to consider both the rectum proper and the pelvic colon. Both portions are liable to be wounded, along with small gut and the bladder. The injury to the small gut is likely to be severe and multiple, though in a few cases it escapes altogether. The lesion of the small gut must be dealt with in the usual way, and if not too extensive, and if no large quantity of blood has been lost, is less to be feared than the wound of the rectum itself. The rectum itself may be wounded extra- or intra-peritoneally, or both. The missiles that cause such injury take a transverse, antero-posterior, or semi-vertical direction (providing the so-called "buttock wound"). If transverse, the entrance and exit wounds lie towards the posterior aspect of the buttocks, and whether the missile catches the extra- or intra-peritoneal surface of the bowel will depend upon the transverse plane traversed and on the obliquity of this plane to the transverse median plane of the body. An important point to remember is that a missile whose track, from a study of the entrance and exit wounds, seems likely to have passed posteriorly to the rectum, has possibly wounded this viscus. Antero-posterior missiles, again, may wound both bladder and rectum. If they are truly antero-posterior, the rectum may be wounded on both surfaces; if somewhat oblique, the intra-peritoneal surface may alone be caught. Again, the pelvic colon may be wounded by a missile traversing the pelvis, such wounds, of course, being intra-peritoneal. I have been struck by the great variations in position of this portion of the bowel, which in some cases may lie on the small intestine in the pelvis, and at other times in the depths of the pelvic basin. The angle at which this loop is perforated also shows that the plane of the loop varies greatly.

Treatment.—There will not be, as a rule, much doubt that an exploratory celiotomy is the right procedure, but if a doubt should arise it is well to be on the side of operation; I refer more particularly to the transverse buttock wounds and the vertical buttock wounds, the last-mentioned having acquired an unenviable reputation in this War. The wounds of the pelvic loop may be small in size, so that suture meets the case. On the other hand, the destruction has sometimes been so great that an iliac artificial anus has been deemed the wisest course. Whether an extensive wound should be repaired depends upon the condition of the patient, and this is not likely to be good if a small intestine lesion has already been dealt with. Intra-peritoneal wounds of the rectum proper are best sutured, though in many cases it seems probable that great difficulty will be experienced, and that a Trendelenburg position is necessary. The question of a transverse colostomy must be determined by the probability of the suture holding, and perhaps is a wise measure in most cases. If a retro-peritoneal wound is present a transverse colostomy with an opening up of the wound is the wisest course. A drain down to the lesion is to be recommended, as an infection is much to be feared; but this, as Sir George Makins has pointed out, is much more likely to form a local abscess than in the case of a small gut lesion. A simultaneous colostomy and a suprapubic drainage of the bladder is to be avoided if possible.

In conclusion, it can be truly said that a fatal result from septic infection is much to be feared although the lesion is small, apparently favourable, and the condition of the patient good. The infectivity of the rectum is well known to all surgeons. Fortunately, rectal wounds do not form a large proportion of abdominal injuries. Among seventy-three cases of great gut wounds there were only six cases of pelvic injury of the rectum proper, and only two of these were sent to the Base. There were, in addition, nine cases of suture of the pelvic colon, and of these five died.

MR. GRAEME ANDERSON, Surgeon, R.N.: Wounds of the rectum have been comparatively uncommon in the present War. Yet curiously enough my first case on going into action at Antwerp was a rectal one, a private of marines receiving what he called "a dose of shrapnel in the stern." During an air raid a bomb dropped from a hostile aeroplane, exploded a few feet from a soldier in a stooping position, blowing off one buttock and causing a charred wound. This case healed remarkably well. Wounds of the pelvic colon do very badly. I suggest that all members of the Sub-Section who are on active service should be communicated with, so that records of, not generalities on, rectal wounds can be kept.

(May 10, 1916.)

Injuries of the Rectum resulting from the War.

By P. LOCKHART MUMMERY, F.R.C.S.

SINCE the beginning of the present War I have seen, in the military hospitals, a number of cases of injury to the rectum and anus from bullet and shell wounds, and I think my experience of such cases may be of interest, especially when it is compared with that of surgeons attending these cases at the Front, for I have naturally seen the patients at a very different stage, and when the indications as regards treatment were quite different. Surgeons at the Front see the cases when the wounds are recent, and when the primary consideration is to save life and control successfully the almost inevitable sepsis. Colonel Cuthbert Wallace's paper is a most valuable contribution to our knowledge from this point of view, and although my experience is not so interesting or valuable as his, it has this advantage: that the cases I have seen were, for the most part, the results of such wounds as he has described. At the time when I saw these wounds it was not so much a question of saving life or controlling sepsis as of dealing with the deformity produced by the healing or part healing of the wounds.

A fact that strikes one particularly in reference to these wounds is that the results are in many cases very crippling. The size of the wounds (particularly where some portion of the bony pelvis has been struck), coupled with the fact that almost without exception the wounds have suppurated violently, results in fearful cicatrization which causes serious stricture and contraction of the parts, more particularly if the anus is involved. One patient whom I was asked to see had been shot through the buttocks and anal canal. The resulting contraction had so closed up the orifice in dense fibrous tissue that colotomy had to be performed. I think that in cases such as this, where serious contraction of the orifice has occurred, the best treatment is a temporary colotomy, followed by an attempt to resect the fibrous tissue and restore some kind of a functional anus at a later date. When the anus is completely destroyed a permanent colotomy probably gives a better functional result than a perineal anus.

One very interesting case which came under my care was that of a soldier who was hit by one of our own shells during an advance. The rectum had been torn open from the anus backwards for about 4 in. When I saw him the wound was healed, but there was a large opening about 3 in. long into the rectum and including the anus—that is to say, the anus formed the extreme front end of the opening. The mucous membrane was, of course, adherent to the skin. The patient had no control, and had been pensioned out of the Army after two unsuccessful attempts had been made to close the opening. I persuaded him to let me make another attempt. First I dissected off the mucous membrane freely for $\frac{3}{4}$ in. all round the wound, together with a fair thickness of tissue. I then sewed up the mucosa with catgut and brought the posterior ends of the sphincter together. Finally, I made two flaps of skin by incisions passing backwards and outwards at an angle of 45° from the central line, and starting at the posterior end of the opening. The flaps so formed were freely undercut till they easily met without tension, and were sewn together in the mid-line. A small drain was put in posteriorly, and another into the rectum through the restored anus. The wound healed well, and the patient left hospital with perfect control about a month after the operation. The secret of a good result in such cases is, I believe, to separate the skin and mucous membrane as much as possible by placing as much tissue between them as can be managed, and to avoid all tension.

No attempt should be made to close wounds of the rectum while there is sepsis. One should wait until healing has occurred before doing any operation to close the opening. I believe, however, that all such openings into the rectum can be closed successfully if sufficient care be taken, always providing that enough of the anal musculature has been left to secure a functional result.

Many bullet wounds of the rectum heal perfectly without causing any serious disability of the rectum, and I have seen cases where a bullet has traversed the rectum from side to side, leaving nothing but a scar which could be felt in the rectal wall. Unfortunately in most cases of this description serious injury to the bladder or other structures in the pelvis has occurred, or sepsis in the surrounding tissues with frequent abscesses gives trouble for months after the rectal wound has healed. I had one case, however, in which the bladder and rectum were shot through from before backwards without any serious results to either organ, and without any extravasation. The bullet had emerged

close to the right sciatic nerve, and the patient's only trouble was from bad sciatica owing to concussion of this nerve.

I have seen several cases of bullets lodged in the sacrum, and so far they do not seem to have caused any serious trouble. I have always advised against any attempt to remove them. In one case a shrapnel bullet had perforated the sacrum from behind and lodged just behind the rectum without damaging it. With a finger in the rectum one could quite easily feel the bullet half buried in the anterior surface of the sacrum.

I was recently asked to see two cases of bleeding from the rectum in men returned sick from the Front, and I think they are worth mentioning. In the first case the history was as follows: The man while at the Front had accidentally fallen into a trench and upon a haversack lying at the bottom, which struck him in the left iliac fossa. He was rather shaken by the fall but soon felt all right. About an hour later, however, he suddenly had a desire to have his bowels opened, and passed a large quantity of blood. After this he passed blood on several occasions and was ultimately invalided home. When I saw him he appeared to be in good health, except that he occasionally passed blood, the last time he did this being about ten days before I saw him. I examined him under an anæsthetic and passed a sigmoidoscope. Nothing whatever could be seen. One could only conclude that there had been some injury to the mucous membrane of the sigmoid from the fall, which had since healed up. In the other case the patient had been invalided back from the trenches on account of bleeding piles. He had been in hospital some two or three weeks and had several times been examined without piles having been discovered, or anything else to account for the bleeding. The hæmorrhage occurred several times a week when he went to stool. It was never much in quantity, and he seemed to be quite normal in other respects. The most careful examination failed to reveal any internal piles, and it was ultimately discovered that the patient was deliberately injuring himself, apparently with his finger-nail, in order to remain on the sick list.

(May 10, 1916.)

Case of Gumma of the Rectum simulating Carcinoma.

By ASLETT BALDWIN, F.R.C.S.

A LADY, aged about 40, injured her coccyx in July, 1896, when jumping off a bicycle which was going quickly, and was treated by her doctor for about six weeks with morphia injections.

She was seen by two surgeons, one of whom diagnosed inoperable carcinoma of the rectum and advised colotomy. The second surgeon saw her in August, 1898. At that time she still suffered acute pain and could not sit down at all—only lie or stand. Carcinoma was again suspected. She was examined under an anæsthetic and an operation done for piles; the coccyx was also removed. She left the nursing home feeling no relief from her pain, and was told by the surgeon she would probably have to return and have more bone removed.

I saw her in October, 1899. She still complained of great pain, and could not sit down properly. On rectal examination, there was a large lump in the posterior wall of the rectum about as far up as the finger could reach; it was fixed to the sacrum, and in its centre was a large and deep circular ulcer. The swelling was smooth and not nodular, and not characteristic of carcinoma. There was no definite specific history, but it was suspicious. The patient was given mercury and iodide, and in three months was quite well and felt better than she had done for years. She took the medicine for several years and still takes it for about six weeks every eighteen months or so. She is now quite well.

(The above history is from a letter just received from the patient, my original notes being lost.)

DISCUSSION.

MR. LOCKHART MUMMERY: Mr. Baldwin's case is one of great interest, as gumma of the rectum is very rare—so rare that hitherto I have only seen one doubtful case. It is most important that every case should be recorded. Mr. Baldwin's case certainly seems to be one of gumma, although absolute proof is wanting. It would be very interesting if Mr. Baldwin would record the subsequent history of the case at some future date.

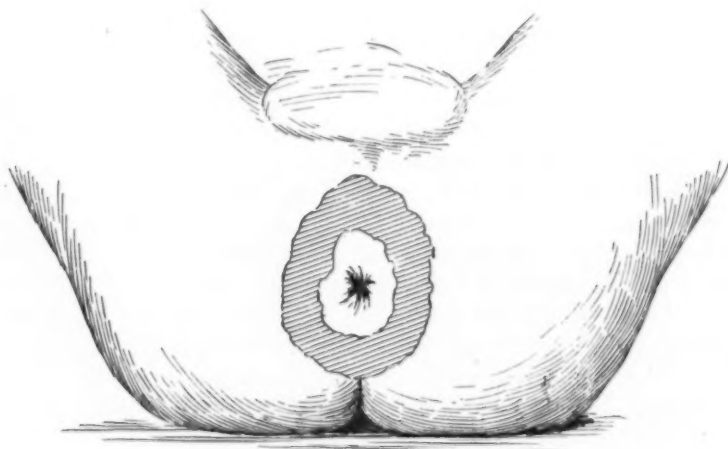
MR. GRAEME ANDERSON, Surgeon, R.N.: There has been a history of injury, probably injury of bone, and a chronic abscess simulating gumma may have resulted.

(May 10, 1916.)

Three Cases of Injury to the Rectum from Bullet Wounds.

By C. GORDON WATSON, Lieutenant-Colonel R.A.M.C.,
C.M.G., F.R.C.S.

THREE cases, all very much alike, were of interest as regards exit wounds in the perineum. The bullets entered from the buttock region or thereabouts. In each case the skin split away from the sphincter margin through its whole circumference, leaving the anus with a small margin of skin isolated (*see illustration*). This was no doubt due to air concussion distending the loose perineal skin, and then bursting like a paper bag, the sphincter holding tight.



Injury to the rectum from bullet wound.

Another point of interest occurred in two cases of perforating wounds of the rectum—viz., considerable blood effusion in the region of the mesentery of the pelvic colon—an argument in favour of transverse rather than iliac colostomy in cases of rectal wounds.

Speaking generally, so far as I have seen, perforating wounds of the rectum tend to do badly because of the severity of the sepsis; and *early* colostomy with thorough exposure of the entire track (when possible) for drainage seems to me to be imperative.

Surgical Section.

President—Surgeon-General Sir ANTHONY A. BOWLBY, K.C.M.G., A.M.S.

(July 21, 1916.)

(Chairman—Sir RICKMAN J. GODLEE, Bart., K.C.V.O., M.S.)

The Treatment of Convalescent Soldiers by Physical Means.

By R. TAIT MCKENZIE, Major R.A.M.C.

THE great military hospital may be compared to a general post office in which the sick and wounded from the front are sorted out into first, second, and third class matter.

The first class matter is distributed rapidly to regular military hospitals and those under the Red Cross, where an operation or a short course of treatment puts them right, and after ten days' leave they go back to the fighting line, with few exceptions.

The second class matter requires an additional stay at a convalescent hospital, commanded by an officer of the Royal Army Medical Corps, in which they receive treatment by physical means, including exercise, in addition to the usual hospital treatment, and a large proportion of these men again find their way to the front.

The third class matter, however—the cases too tedious for the hospital and the convalescent camp—are more difficult to provide for and dispose of. During the first year of the War they were referred from depot to depot, always finding that they were at the wrong address, and it was not until last autumn that the Director-General was able to give his attention to these cases that had begun to accumulate in considerable numbers. He arranged a series of Command Depots, commanded by a combatant officer for discipline, with medical officers

attached, and to them have been sent all for whom there was some hope of cure, or improvement, within a period of six months.

The objects of these depots were to return every available man to active service by treatment; to return men fit for light service abroad who could replace fit men in light duties on lines of communication; to fill positions requiring light duty at home by men who were unable to do anything more than to release a better man for active service, and to discharge from the Army those for whom no treatment could be expected to give further results.

These Command Depots afforded welcome relief to the various regimental depots and other places overcrowded with men useless from



FIG. 1.

Treatment of two arms at once by galvanism in the Schnee bath.

the military standpoint, and of but little use to themselves—men who were rapidly lapsing into complete or partial invalidism, and whose deterioration from a military standpoint was even greater, under the combined influence of sympathy and freedom from all duty.

Either two or four thousand men constitute the complement of patients for a Command Depot—a strange assemblage of cases: profound neurasthenia, the result of sleepless nights and arduous days; shock in all its forms—tremulous hands and tongue, stammering speech or deafness, persistent nightmares, and fears by day; disorders of sensation, contractures and paralyses; rapid and weak heart action, hearts that were overstrained and that are unable to sustain the effort of the

lightest gymnastic exercise or the shortest march; rheumatism, real and unreal, in all its forms; lungs suffering from the bronchitis of gas poisoning, asthma and even tuberculosis; profound debilities following typhoid, dysentery and malaria, requiring months of good food, light duty and progressive exercise to build them up. Then the wounded—an endless stream—feet and legs pierced by bullets, or pitted by shrapnel, arms and hands with torn nerves and jagged tender scars; chests still containing bullets or pieces of shell; in fact no part of the body escapes the awful and sometimes fantastic effects of the ordeal through which it has passed.



FIG. 2.

Ionisation with potassium iodide solution. The "negative" electrode, composed of copper wire mesh, covered by felt, extending from back to heel; the "positive" electrode, on front of thigh and leg, held in place by sandbags.

For almost all these cases the treatment comes under what might be called "physical therapy"—electricity, hydro-therapy, massage, mechano-therapy, corrective exercises, physical training and marching.

The electrical equipment consists of the three currents usually supplied by the polystat. The faradic is used largely in diagnosis of nerve injury, in the treatment of functional conditions of the heart and for the stimulation of wasted muscles. By the use of the condenser the current is rhythmically interrupted and varied in intensity, and a good substitute for exercise is obtained where movements cannot be made (fig. 1). The galvanic current, transmitted through water in the Schnee arm and leg baths is useful for weakened limbs (fig. 1), for



FIG. 3.

Hydrotherapeutic hut and boiler house, Heaton Park, Manchester.



FIG. 4.

The whirlpool baths for arms and legs.



FIG. 5.

Scar that would break down if soaked in water, but which requires radiant heat.



FIG. 6.

Ankylosis of elbow, wrist and fingers, with muscle wasting due to long confinement in splints; complete recovery under massage and stretching.

certain cases of frost-bite, and other conditions of impaired circulation, and for the softening of scar tissue by ionization with sodium chloride and iodide of potassium solutions (fig. 2), while the sinusoidal current, less irritating than the faradic current, is of value in keeping up the nutrition of muscles whose nerve supply is defective. The ordinary electrodes do not serve for these cases and we have used copper-wire mesh, cut to fit the front of the thigh, the back of the leg, or any other special region. They are covered with felt after the terminals have been soldered on, and are inexpensive.

The Committee on Balneology of the Balneological and Climatological Section of the Royal Society of Medicine gave the War Office



FIG. 7.

Blind soldier as masseur.

the benefit of their experience in planning an ideal equipment for hydrotherapy, by which the ordinary hut is turned into a complete hydro with the douche, tub bath, a pool and whirlpool baths (fig. 3, exterior of hut).

The stimulating douche, hot and cold, has been of use not only in the treatment, but also in the diagnosis of the many cases of rheumatism, two or three treatments sometimes being sufficient to cause an apparently confirmed cripple to declare himself quite able to undertake full physical training, other cases, where the diagnosis was not in doubt, being progressively improved. Many of these cases are treated daily in

the tub bath—fifteen to twenty minutes, with the temperature at 98° F., followed by general massage.

The pool is kept at a constant subthermal temperature, about 94° F. It is large enough to contain twelve men sitting up to their neck in water, in which they remain for an hour, and there is a constant succession of patients. It is now applied as a routine treatment for practically all cases suffering from shock and disordered heart action. Under its influence a rapid and excited pulse goes down and an hour's rest continues this sedative effect.

In the whirlpool bath (fig. 4) limbs with painful scars, or frost-bitten feet, are submerged in water at 110° F., violently agitated by a continuous flow from two or three jets, or still better by a turbine in the



FIG. 8.

Passive flexion of ankle-joint by manipulation.

bottom. The introduction of compressed air into the stream turns it into a bubbling and effervescent envelope for the painful arm or leg. It enables one to apply more heat and stimulation, with water at a lower temperature than would be possible if the water were still. After twenty minutes' immersion, the whole part is flushed with blood, the pale limb and purple scar become crimson and comfortable, and the circulation remains heightened for hours. It makes an ideal preparation for the necessary massage and manipulation that would not be tolerated without it.

In many cases, however, it is not advisable to have a scar sodden by water (fig. 5), so that the heat must be applied in a dry form. For this the radiant light baths are designed, and need not be complicated or

expensive. We have found the small hinged shoulder light fulfils all the requirements. It can be adjusted to any single joint at will, and a row of three or four can be placed over the back from the hip to neck, covered by a blanket, and in twenty minutes the whole region so covered will be flushed and reddened, making an excellent preparation for the massage that usually follows. In the great majority of cases at a Command Depot, electricity, hydrotherapy and radiant light are but preparations for massage.

The Almeric-Paget Massage Corps has supplied trained masseuses, one operator being able to treat twenty cases a day, and recently the

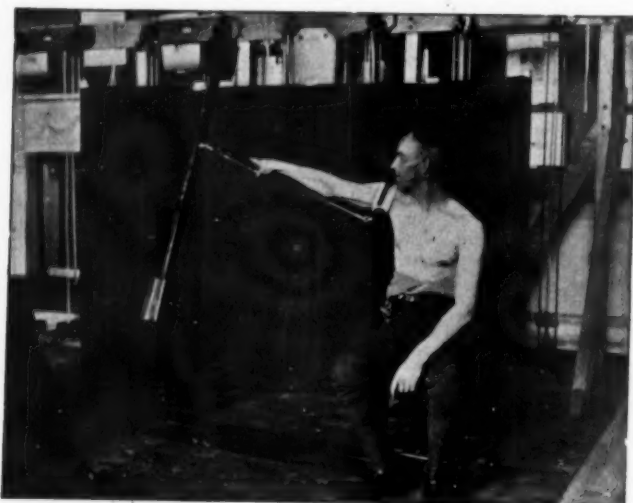


FIG. 9.

Circumduction of the shoulder, active or passive.

Corps has been strengthened by the first graduates from St. Dunstan's—four men blinded in action and taught their profession since the beginning of the War. The histories of cases neglected for months, or maltreated by unskilled amateurs, show the imperative necessity for careful training in this most important work (fig. 7).

Many cases come in with limbs which have become wasted and joints stiffened by long immobility in splints (fig. 6), cases which must be slowly coaxed back to strength and usefulness by skilled massage and

manipulation, and other apprehensive patients appear on inspection whose joints have been rendered painful, and in whom freshly formed scar tissue has been torn and inflamed by manipulations which a well trained masseuse would never have undertaken.

As massage progresses it is followed by passive manipulation, either by the hand of the operator (fig. 8) or by machines designed to stretch shortened ligaments, break down slight adhesions, and restore the normal movement in joints.

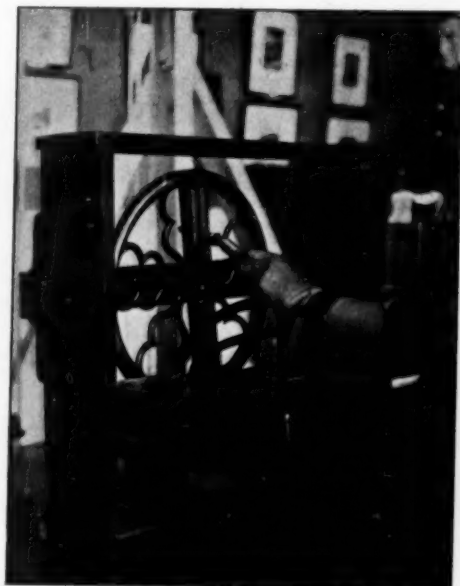


FIG. 10.

Circumduction of the wrist, active or passive.

The Zander machines for passive movement are quite beyond the reach of any but the very few owing to the cost. They are unnecessarily complicated, but substitutes giving the same movements have been designed which will produce the same effects at a cost of as many shillings as the Zander machines cost pounds. Circumduction of the shoulder, with increasing range both active and passive, is given by one inexpensive machine (fig. 9) made of two uprights of wood, one with crutch-like support for the shoulder and the other having attached to it

an arm with movable weights—the two connected by a jointed rod grasped by the patient, and capable of being moved out or in at the will of the operator. By it we can progressively increase the range of movement in the shoulder. Another acts on the wrist (fig. 10) and still another on the ankle (fig. 11).

Treatment must, however, not stop at passive manipulation, for the muscles and joints must be systematically re-educated to take up again their normal movements, and again the expensive and cumbrous Zander machine can be replaced by simple devices to strengthen enfeebled muscles by giving them definite tasks of increasing difficulty. Specially



FIG. 11.

Passive flexion of ankle-joint by swinging a counter-weight.

designed machines are now in use at Heaton Park in which the principle of the weight and pulley is employed. Flexion and extension of the fingers at the metacarpo-phalangeal joints and at the interphalangeal joints can be obtained by one (figs. 13 and 14) having a wire hoop with rings through which cords are threaded, and attached to a cotton glove by one end, the other end terminating on a ring to which are hung shot-bags weighing 2, 4, 6, and 8 oz., so that the muscles can be given an increasing load as they become stronger. The direction of the pull is changed so as to obtain flexion or extension by adjusting the hoop. Other devices are used for the wrist (fig. 15), elbow, shoulder, trunk,

thigh (fig. 12), leg and foot; but to be effective movements on them must be given with great accuracy and in known doses (fig. 16). It is easy to mistake the real action that has been performed on one of these machines; the normal action of a stiffened wrist is simulated by lifting the elbow, and what looks like pronation may be in reality a rotation of the shoulder.

Many cases of functional paralysis in men who have been crushed or buried have been treated successfully by these means, one extreme case of paralysis of the abdominal muscles making a quick and complete recovery (fig. 17).

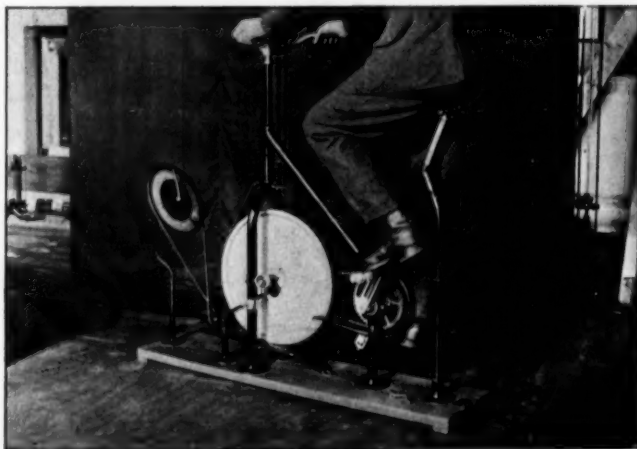


FIG. 12.

Bicycle trainer for thigh and calf muscles.

When a patient is able to perform these movements he must be thrown still more on his own resources by free gymnastics in which there is no guidance from the machine or operator, and cases are then sent to the gymnastic instructor, who begins with the three tables of carefully compiled exercises, starting with half-an-hour's repetition of such simple movements as raising the heels (fig. 18), turning the head, and bending the arms and trunk; going over the body region by region in a definite progression (fig. 19). Here the wounded man is joined by cases of shock who are able to do only the lightest exercise, by men whom neurasthenia, enteric or dysentery has left profoundly

debilitated, and slowly they progress through this light physical training from stage to stage until they are able to march in quick time, to bend and twist, and use their arms freely (fig. 20). When they are able to do all these movements they are promoted to full physical training, which introduces quick and active movements, running, jumping and gymnastic games, teaching alertness, control and agility, walking balanced on raised beams (fig. 21), or lifting their feet high



FIG. 13.

Active flexion of fingers at metacarpo-phalangeal joints by raising shot-bags on cords attached to glove fingers and passing through rings.

as they go through wire entanglements (fig. 22), climbing walls and leaping ditches. Parallel with this course goes graduated walking on level and then on hilly ground, starting with a mile or two at the patient's own pace, and even with the help of a stick, progressing to three or four miles, and graduating finally into the full route march at quick time.

The patient who can do his full physical training and full route march is ready for the final stage of hardening in which he does the full table of exercise required of the trained soldier, and a twelve or fifteen mile march in full kit. This test is necessary, for sometimes it is only when the kit is put on that the healed scar or old fracture becomes painful (fig. 23).

Many cases of disordered heart action can be brought up to the full physical training, but not into it. After the first day of it their pulse,



FIG. 14.

Active extension of metacarpo-phalangeal joints, showing the adjustment of the hoop and rings for this movement.

which has been brought down to a normal of 80 by faradism or sub-thermal baths and rest, and which remains undisturbed after a gentle slow walk, jumps up to 120 or 140, and can only be reduced by a reduction of the amount of work required. Several cases were brought up to this point on three or four occasions and had to be returned to

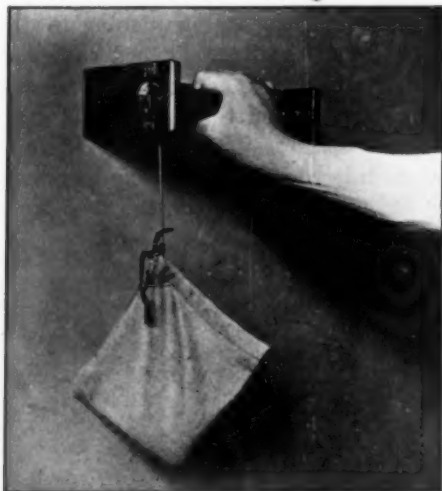


FIG. 15.

Flexion of wrist by roller and weights. Each end of the roller is notched to hold the weight at the end of flexion or extension.



FIG. 16.

Exercises given by prescription for thigh, shoulder, forearm and chest, by pulley weights, at Heaton Park, Manchester.

lighter duty. Not more than 30 per cent. succeeded in passing into Class A. This experience has also been shared by some cases of shock. Bronchitis, following exposure to gas, is frequently re-awakened by the forced effort of running or jumping, and many cases of frostbite, that are quite able to walk a mile or two, find themselves unequal to a forced march and have to be left on light duty.

We shall in future be able to refer suitable cases to the special hospitals now in course of organization for the treatment of difficult



FIG. 17.

Paralysis of abdominal muscles after being buried in a trench, with complete recovery after re-education of muscles.

orthopædic cases, obscure and puzzling nervous conditions, and for the thorough and minute study of the heart, where individual attention can be paid to cases that would otherwise have to be taken in groups.

A glance at the final result obtained in a Command Depot, will, I think, show how thoroughly justified the Director-General has been



FIG. 18.

Heels raising by the aid of sticks—one of the first exercises for crippled men.



FIG. 19.

Knee-raising by a squad on light physical training.

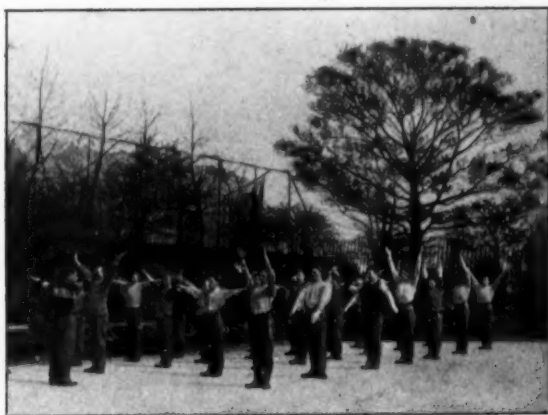


FIG. 20.

Slow deep breathing with arms raising, each man judging his own time.



FIG. 21.

Balance exercises for men in Class "A."



FIG. 22.

High knees raising through wire entanglements for men in Class "A."



FIG. 23.

Powerful man who cannot carry a pack on account of fractured clavicle and scar on the shoulder.

in establishing them in spite of the difficulties met with in manning and equipping these great camps.

At Heaton Park an analysis of all classified cases sent out up to date show that out of all men discharged nearly 50 per cent. have been rendered fit for active service, and have rejoined their units in the fighting line; about 15 per cent. have been sent to lines of communication abroad; 15 per cent. have been sent to useful work of a sedentary character at home; and 20 per cent. have been discharged as "permanently unfit," many of these being untreatable from the first.

The average time of each man spent in treatment was well under three months, and the fact must not be lost sight of that even though a man is not sent back in category "A" his opportunities for a useful career in civil life after the War have been enormously increased, and the burden on the Nation in future pensions correspondingly lightened.

Note.—I wish here to acknowledge the assistance of Captain A. E. Barclay, R.A.M.C., whose help in time, ideas and money made it possible to make the experimental machines for corrective exercises. I am also indebted to Dr. H. J. Seeuwen, for notes on cases under his immediate charge for electrical treatment, and to Dr. Frank Ratcliffe for special and valuable assistance in observing cases under treatment by hydrotherapy.

APPENDIX A.

ANALYSIS OF THE FIRST 1,000 CASES.

Nerve injuries, shock, &c.	71
Heart	54
Rheumatism	90
Lungs: Bronchitis, tuberculosis, asthma	67
Debility following typhoid, malaria, and dysentery	151
Wounds of lower limbs	206
Wounds of upper limbs	179
Wounds of trunk	215
Wounds of head	79
Diseases of eyes	27
Diseases of ears	40
Diseases of teeth	31

TREATMENT ADVISED.

Massage	313
Electricity	87
Hydrotherapy	321
Remedial exercises	237
Light route marches and light physical training	615
Full physical training, including full route march (6 miles)	133
Unsuitable for treatment	200
Returned to unit	66
Returned fit without treatment	41

APPENDIX B.

GUIDE TO OFFICERS AND CIVILIAN PRACTITIONERS IN MAKING MEDICAL EXAMINATIONS OF
NEW ARRIVALS, AND IN ALL RE-EXAMINATIONS.

Treatment	Conditions for which they are prescribed	Abbreviations
(1) <i>Massage</i> ...	(1) Contracted scars, (2) stiffened joints, (3) nerve injuries, (4) paralysis, (5) certain rheumatic conditions, (6) trench feet	
(2) <i>Radiant Heat</i> ...	(1) Painful scars, (2) painful rheumatic joints and muscles	R. Heat
(3) <i>Electricity</i> : (a) faradic, (b) galvanic, (c) sinusoidal, (d) ionization	(1) Painful scars, (2) paralysis, (3) disordered action of the heart, (4) contractures	Elec.
(4) <i>Vibration</i> ...	(1) Disordered action of the heart, (2) rheumatism of back and thigh	
(5) <i>Corrective Exercises</i> ...	(1) Stiffness or weakness of fingers, (2) wrist, (3) elbow, (4) shoulder, (5) back, (6) abdomen, (7) thigh, (8) calf, (9) ankle, (10) foot	Med. exercises
Remedial exercises (in small classes)	(1) Flat foot, (2) bronchitis after gas poisoning, (3) gunshot wounds in chest	Med. exercises
(6) <i>Hydrotherapy</i> : (a) douche, (b) sedative bath, (c) whirlpool bath, (d) brine bath	(1) Nervous shock, (2) rheumatism, (3) disordered action of the heart, (4) painful joints, (5) contractures, (6) trench feet	Hydro.
(7) <i>Hospital Treatment</i> ...	(1) Medical, surgical, and dental, (2) eye conditions, (3) ear conditions, (4) sputum examinations, (5) X-ray examinations	Reports required
(8) <i>Light Route March</i> : two to four miles' free walking	(1) Debility after enteric and dysentery, (2) partial recovery from wounds	L. R. M.
(9) <i>Light Physical Training</i> : movements slow and without running or jumping	(1) Debility, (2) partial recovery from wounds	L. P. T.
(10) <i>Full Physical Training</i> : including running and jumping and gymnastic exercises, also Full Route March in quick time	For men ready for Class "A" and preparing to return to their unit	F. P. T. F. R. M.

APPENDIX C.

CLASSIFICATIONS AS PER ARMY COUNCIL INSTRUCTION No. 1,023 OF 1916.

Categories	Sub-heads
(A) <i>Fit for general service</i> ...	(i) Men able to march, see to shoot, hear well, and stand active service conditions
(B) <i>Fit for service abroad but not fit for general service</i>	Men free from serious organic disease, able to stand service conditions on the line of communications in France, or in garrisons in the Tropics, also : (i) Able to march at least five miles, see to shoot with glasses, and hear well (ii) Able to walk to and from work a distance not exceeding five miles, see and hear sufficiently for ordinary purposes (iii) Only suitable for sedentary work
(C) <i>Fit for service at home only</i> ...	Men free from serious organic disease, able to stand service conditions at home, also : (i) Able to march at least five miles, see to shoot with glasses, and hear well (ii) Able to walk to and from work a distance not exceeding five miles, see and hear sufficiently for ordinary purposes (iii) Only suitable for sedentary work
(D) <i>Temporarily unfit for service in categories A, B, or C, but likely to become fit within six months</i>	(i) Men in this category should all be under treatment and re-classified when as much improvement as possible is obtained
(E) <i>Unfit for service in categories A, B, or C, and not likely to become fit within six months</i>	(i) Awaiting discharge or re-classification

APPENDIX D.

PRESCRIPTIONS OF REMEDIAL EXERCISES AS USED AT HEATON PARK.

Figures refer to illustrations in text-book, "Exercise in Education and Medicine" (W. B. Saunders and Co.). Weights raised by pulleys should be gradually increased and number of times up to twenty for each movement.

(I) REMEDIAL EXERCISES : FOREARMS AND WRISTS.

(A) *Movement with Resistance.*

- (1) Flexion ; extension of elbow with resistance. (See "Exercise in Education and Medicine," figs. 423, 424.)
- (2) Fist clenched ; flexion, extension, adduction and abduction of the wrist with resistance.
- (3) Pronation and supination with resistance.

(B) Pulley Weights.

- (1) Position : Face to, arms downward ; floor attachment.
Movement : Flex and extend elbow.
- (2) Position : Face from, arms forward, palms up ; high attachment.
Movement : Flex and extend elbow.
- (3) Position : Face from, arms downward ; floor attachment.
Movement : Flex and extend elbow.
- (4) Position : Face to, arms downward, elbows bent ; high attachment.
Movement : Extend and flex elbow.

(C) Wrist Machines.

- (1) Handle Grip : Pronation and supination with increasing weights.
- (2) Roller Grip : Overhand, winding movement.
- (3) Roller Grip : Underhand, winding movement.
- (4) Circumduction of wrist, passive and active on machine.

(II) REMEDIAL EXERCISES : SINGLE SHOULDER.

(A) Movement with Resistance.

- (1) Arm raise side upward with resistance.
 - (2) Arm lower side downward with resistance.
 - (3) Arm raise forward with resistance.
 - (4) Arm raise backward with resistance.
 - (5) Arm circumduct with resistance.
- (See "Exercise in Education and Medicine," figs. 421 to 430).

(B) Pulley Weights.

- (1) Position : Side to, arm sideward raised ; high attachment.
Movement : Arm forward across chest, elbow straight.
- (2) Position : Side to, arm sideward raised ; floor attachment.
Movement : Arm upward raise, elbow straight.
- (3) Position : Side to, arm sideward raised ; high attachment.
Movement : Arm downward and forward, arm downward and backward, alternating.
- (4) Position : Side from, arm across chest ; high attachment.
Movement : Arm side outward, elbow straight.
- (5) Position : Side from, arm downward and behind ; floor attachment.
Movement : Arm side upward raise.

(C) Shoulder Machine.

Stretching contractures up to point of pain. (If pain persists discontinue and report.)

(III) REMEDIAL EXERCISES : CHEST AND SHOULDERS.

(A) Free.

- (1) Arms forward raise, inhale, side downward lower, exhale. (See "Exercise in Education and Medicine," figs. 304, 305.)
- (2) Arms side upward raise, inhale, trunk forward bend and raise, arms side downward lower, exhale. (See "Exercise in Education and Medicine," figs. 309, 310.)

(B) Pulley Weights.

- (1) Position: Face to, both arms forward; high attachment.
Movement: Arms downward, arms outward, arms upward, repeating in rotation.
- (2) Position: Face from, arms forward, elbows straight; high attachment.
Movement: Arms flex, inhale; arms forward, exhale.
- (3) Position: Face from, arms downward; floor attachment.
Movement: Arms forward raise, elbows straight.
- (4) Position: Face from, arms forward; high attachment.
Movement: Arms raise, inhale; arms lower, exhale.
- (5) Position: Face from, arms forward; high attachment.
Movement: Arms sideward and inhale; forward, exhale.

(C) Stall Bars.

- (1) Span bending with chest raising one or both arms. (See "Exercise in Education and Medicine," fig. 521.)
- (2) *For one side only.* Overhead grasp and side stretching.

(D) Plint.

- (1) Supine. Artificial respiration with slow, deep breathing. (See "Exercise in Education and Medicine," figs. 411, 412.)
- (2) Supine. Slow breathing with arms forward upward raising and side downward lowering. (See "Exercise in Education and Medicine," fig. 312.)

(IV) REMEDIAL EXERCISES: ABDOMINAL MUSCLES.

(A) Free.

- (1) Position: Standing, hands clasped across abdomen.
Movement: Abdominal breathing. (See "Exercise in Education and Medicine," fig. 401.)
- (2) Position: Standing, hips firm.
Movement: Trunk circumduction. (See "Exercise in Education and Medicine," figs. 391, 392.)

(B) Stall Bars.

- (1) Position: Sitting on stool, hips firm, feet fixed.
Movement: Trunk extension and flexion, with twisting. (See "Exercise in Education and Medicine," fig. 393.)
- (2) Position: Hanging to stall bars, face outwards.
Movement: Raise knees, extend and flex legs with knees raised.
- (3) Position: Standing, back to.
Movement: Span bending and chest raising. (See "Exercise in Education and Medicine," fig. 52.)

(C) Plint.

- (1) Position: Supine, hips firm.
Movement: Both legs raise 12 inches, knees straight, alternate feet raise and lower 6 inches, ten times; legs lower. (See "Exercise in Education and Medicine," fig. 450.)
- (2) Position: Supine, hips firm.
Movement: Trunk raising and lowering. (See "Exercise in Education and Medicine," fig. 452.)
- (3) Position: Sitting astride, neck firm.
Movement: Trunk circumduction. (See "Exercise in Education and Medicine," Plate iii, fig. 2.)

(V) REMEDIAL EXERCISES: SHOULDERS AND BACK.

(A) *Free.*

- (1) Arms forward raise, inhale, side downward lower, exhale. (See "Exercise in Education and Medicine," figs. 304, 305.)
- (2) Arms side upward raise, inhale; trunk forward bend and raise; arms side downward lower, exhale. (See "Exercise in Education and Medicine," figs. 309, 310.)

(B) *Pulley Weights.*

- (1) Position: Face to, arms forward; floor attachment.
Movement: Both arms upward stretch.
- (2) Position: Face to, arms forward; floor attachment.
Movement: Forearms flex and extend, drawing the elbows back.
- (3) Position: Sitting on rowing machine.
Movement: Rowing movement with increasing weights.

(C) *Plint.*

- (1) Position: Prone, neck firm, feet fixed.
Movement: Extend back and neck. (See "Exercise in Education and Medicine," fig. 311.)
- (2) Position: Sitting astride, neck firm.
Movement: Flexion and extension of trunk.
- (3) Position: Prone, arms bend, feet fixed.
Movement: Swimming. (See "Exercise in Education and Medicine," figs. 313 to 315.)

(VI) REMEDIAL EXERCISES: THIGH.

(A) *Movements with Resistance.*

Position: Standing with support for hand.

Movement: Thigh abduction, adduction, flexion and extension with resistance.

(See "Exercise in Education and Medicine," figs. 438, 439, 440, 441, 443.)

(B) *Pulley Weights.*

- (1) Position: Side to, strap to foot; floor attachment.
Movement: Hip adduction, knee straight.
- (2) Position: Face to, strap to foot; floor attachment.
Movement: Hip extension, knee straight.
- (3) Position: Side from, strap to foot; floor attachment.
Movement: Hip abduction, knee straight.
- (4) Position: Face from, strap to foot; floor attachment.
Movement: Hip flexion, knee straight.

(C) *Bicycle Trainer.*

Increasing distance and friction.

(VII) REMEDIAL EXERCISES: LEG AND FOOT.

(A) *Movements with Resistance.*

- (1) Position: Standing, knee flex and extend. (See "Exercise in Education and Medicine," figs. 442, 443.)
- (2) Position: Sitting, flex and extend, adduct, abduct foot with resistance. (See "Exercise in Education and Medicine," fig. 446 and figs. 442, 443.)
- (3) Position: Standing, toes in.
Movement: Heels raise, full knees bend.

(B) Pulley Weights.

- (1) Position : Face to, strap to foot ; floor attachment.
Movement : Flex knee.
- (2) Position : Face from, strap to foot, knee flexed ; floor attachment.
Movement : Extend knee.

(C) Bicycle Trainer.

- (1) Increasing distance and friction.
- (2) Circumduction of the ankle, passive or active on machine up to the point of pain.

APPENDIX E.

NOTES ON TABLES OF EXERCISES.

These four tables of exercises are to control the training of convalescents.

Duration of table about half-an-hour, repeated daily for twelve to eighteen days.

In moving from one table to another after teaching the new marching exercise, the marching exercise of the previous table or tables should be frequently practised.

Occasionally high and long jumping should be substituted for jumping and vaulting exercises.

Games should occasionally be substituted for marching and jumping and vaulting exercises on reaching Tables II and III, such as relay races, front rank v. rear rank, under passing relay race, jumping the bag, three deep (or "tag"), cat and the mouse, slipper tag from a circle.

Quickening exercises, either those of a rhythmical or competitive nature, should be applied at any time when a slackening of attention occurs.

The nomenclature of positions and exercises is that authorized and approved by the Headquarters Gymnastic Staff at Aldershot, and the tables can be taught by any of their certificated instructors.

FIRST TABLE OF EXERCISES IN LIGHT PHYSICAL TRAINING.

(A) Introductory Exercises.

Leg exercise	(a) Feet close ; (b) heels raise.
Neck exercise	Head backward bend.
Arm exercise	(a) Hips firm ; (b) arms bend ; (c) arms bend, arms upward stretch.
Trunk exercise	Feet close, hips firm, trunk turning.
Leg exercise	(a) Hips firm, foot sideways place ; (b) feet astride place.

(B) General Exercises.

Preparation for span bending	Feet astride, arms upward stretch (taken free).
Balancing exercise	Hips firm, knee raise.
Lateral exercise	Feet close, hips firm, trunk bending sideways.
Abdominal exercise	Hips firm, feet sideways place, trunk bending backwards.
Dorsal exercise	Hips firm, feet sideways place, trunk bending forwards.
Marching exercise	Quick march ; (later) on the toes march.

Practice class arrangements : in two ranks fall in, numbering, opening and closing ranks.

(C) *Final Exercises.*

Heels raise. Arms raising sideways.

SECOND TABLE OF EXERCISES IN LIGHT PHYSICAL TRAINING.

(A) *Introductory Exercises.*

Leg exercise	Hips firm, heels raising and knees bending (later quickly).
Neck exercise	Head bending backward (later head turning).
Arm exercise	(a) Arms bend, one arm upward one arm downward stretch; (b) arms bend, arms sideways stretch.
Trunk exercise	Hips firm, foot sideways place, trunk turning.
Leg exercise	(a) Feet close and full open; (b) hips firm, foot placing sideways.

(B) *General Exercises.*

Preparation for span bending	Feet astride, arms upward stretch, trunk backward bend (slight movement only).
Balancing exercise	Hips firm, leg raising sideways; (later) "backward" and "forward."
Lateral exercise	Hips firm, foot sideways place, trunk bending sideways; (later) feet close, one arm downwards, trunk bending sideways.
Abdominal exercise	(a) On the hands down (by numbers); (b) on the back down (ground permitting); (later) with leg raising.
Dorsal exercise	Hips firm, foot sideways place, trunk forward bend, trunk downward bend.
Marching exercise	Hips firm, with knee raising quick mark time.
Jumping and vaulting exercise	Upward jumping.

(C) *Final Exercises.*

Hips firm, foot sideways place, trunk turning. Heels raise. Arms raising sideways and upwards.

THIRD TABLE OF EXERCISES IN LIGHT PHYSICAL TRAINING.

(A) *Introductory Exercises.*

Leg exercise	(a) Arms bend, heels raise, knees bend, arms stretching sideways; (b) hips firm, feet full open, heels raise and full knees bend.
Neck exercise	Head turning quickly (later head bending forward).
Arm exercise	(a) Arms sideways raise, arms forward bend; (b) arms swinging upward.
Trunk exercise	Arms bend, foot sideways place, trunk turning.
Leg exercise	(a) Hips firm, foot placing sideways; (later bend); (b) hips firm, feet full open, foot outward place; (later) foot placing outwards.

(B) *General Exercises.*

Span bending	Feet astride, arms upward stretch, trunk backward bend.
Complex exercise	Hips firm, feet sideways place, trunk bending forward and full downward.
Balancing exercise	Hips firm, leg raising forward, sideways, and backward.
Lateral exercise	Arms bend, feet sideways place, trunk bending sideways.
Abdominal exercise	(a) On the hands down (by numbers); (b) on the back down, leg raising.

Dorsal exercise	Hips firm, feet sideways place, trunk bending forward and downward.
Marching exercise	Hips firm, in quick time sideways march (later slow march).
Jumping and vaulting exercise	With three paces forward, off the left or right foot jump.

(C) *Final Exercises.*

Hips firm, feet sideways place, and trunk turning. Hips firm, foot placing sideways.
Arms raising forward and upward, lowering sideways and downward.

TABLE OF EXERCISES IN FULL PHYSICAL TRAINING.

(A) *Introductory Exercises.*

Leg exercise	Hips firm, feet full open, heels raising and full knee bending (four times).
Neck exercise	(a) Head bending backward (twice); or (b) head turning (twice in each direction).
Arm exercise	Arms bend, arms stretching forward, sideways, and upward (three times).
Trunk exercise	Arms bend, feet sideways place, trunk turning (twice in each direction).
Leg exercise	Hips firm, feet full open, outward lunging (twice each foot).

(B) *General Exercises.*

Span bending	Feet astride, arms upward stretch, trunk bending backward (three times).
Complementary exercise	Feet astride, arms upward stretch, trunk bending forward and full downward (twice).
Supplementary exercise	Hips firm, heels raising and knees bending (three times).
Balancing exercise	(a) Hips firm, leg raising forward, sideways, and backward (twice each); (b) hips firm, knee raising, leg stretching forward (twice each leg).
Lateral exercise	(a) Feet closed, one arm upward one arm downward stretch, trunk bending sideways (twice to each side).
Abdominal exercise	On the hands, arms bend (three times), and add later with leg raising (twice with each leg); or (ground permitting) lying arms upward raising, legs raising (three times).
Dorsal exercise	Arms bend, feet sideways place, trunk forward bend, arms stretching sideways (three times).
Marching exercise	Quick march, double march, marching on the toes (to be done each time). Slow march: Hips firm, with knee raising quick march (one to be done each time). Double mark time: With hips firm, knee raise. Quick march: With hips firm, on alternate feet hop.
Jumping and vaulting exercise	Complete training on obstacle course.

(C) *Final Exercises.*

Leg exercise	Hips firm, heels raising and knees bending (four times).
Trunk exercise	Hips firm, feet close, trunk turning (twice each way).
Corrective exercise	Arms raising forward and upward lowering sideways and downward (until the action of the heart and lungs is eased).

Note.—Duration of a table, including obstacle course, about one hour.

APPENDIX F.

OBSERVATIONS ON HEART CONDITIONS BY H. J. SEEUWEN, M.D.¹

In January last, at a meeting of the Royal Society of Medicine, Sir James Mackenzie opened a discussion on the "Soldier's Heart." His paper was most interesting, as was the discussion which followed his reading. A few weeks afterwards Dr. R. M. Wilson, his assistant, paid a visit to our Command Depot at Heaton Park. He examined a great number of our men affected by heart troubles.

After his visit, Major Tait McKenzie arranged for me to give electrical treatment to a selected group of those men. I took some sixty of the most serious cases of heart troubles. All these men were at the same time given daily open air exercises by Major McKenzie's method, which included gymnastic training and route marches. The physical training includes two divisions: the light and the full training. The first consists of slow movements of limbs and trunk, and some deep breathing exercises; in the second division we find all the movements much more vigorous, much quicker, and also running and jumping.

For the route marches we have the light one, which is only a walk at an easy pace for about two miles; the full route march is a sharp walk, in quick time, for half an hour to an hour, usually with a band of music. I made a daily application with the faradic current, the only form of medical electricity at our disposal for the time being (March, 1916).

The history of most of those men corresponds closely with those related in the articles of Mackenzie, Wilson, and others. Wounds (20 per cent. were wounded), shell shock, excessive work, exhaustion, and nervous strain are common factors. Debility following infection by sore throats, measles, dysentery, typhoid, malaria, were found in about 25 per cent.

When I carefully scrutinized the pathological history of all those young men, in more than 50 per cent. I could trace some old-standing lesions, or some weakness, or at least a want of energy on the part of the heart.

Some of them had suffered years before they enlisted from palpitation, or breathlessness, after any strong effort, such as a run, or

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emotional disturbance; others had been suffering from attacks of rheumatism (with or without heart troubles); others had hyperthyroidism; others had suffered habitually, in winter, from cold hands and feet, or other vasomotor disturbances.

I asked all those men if they could play football or other exhausting games before they enlisted, and found that only five or six out of the sixty men were athletes.

Another very important factor is the probability of infection from bad teeth. Out of those sixty men only eight presented a healthy denture. Is this not one way in which we must expect to discover a very frequent cause not only of the "soldier's heart," but also of rheumatism, or what is commonly called rheumatism, of dysentery, typhoid, goitre, and many other infections which find a well-prepared soil in bodies in which digestive troubles have arisen through defective teeth. In such cases the alimentary tract is the first affected but afterwards the central nervous system suffers, and all kinds of nervous symptoms may well be produced.

Dr. Wilson called my attention to the great number of enlarged necks in the men he examined. The thyroid gland is very often hypertrophied (25 per cent. had an enlarged neck). Is this due to infection and auto-intoxication? In a great number of cases, I think, it is.

During some ten months I saw with Dr. A. E. Barclay in the X-ray Department at the Manchester Royal Infirmary, a great number of cases of simple and exophthalmic goitres, and we found very few of those patients—nearly all women—with a healthy denture.

With regard to indulgence in spirits and tobacco, about which Dr. Poynton spoke, I think that in a few cases this may be a factor, especially as regards the tobacco used in cigarettes where smoke is inhaled. Some of these boys were smoking and drinking more than they did before they were soldiers. Nevertheless only five out of the sixty men were smoking over an average of ten cigarettes per day; most of them smoked only half that quantity.

The following are the results obtained after two months of the above-mentioned treatment. All the men were put as soon as possible on daily light physical training and the light route march. Most of them could stand both those exercises completely or partially, the duration of which was half an hour each, as will be seen from the following case:—

Private J. had dysentery in Gallipoli. He was invalided on October 15; was in hospital for three and a half months, and came into camp at the end of

February. He was short-winded and had palpitations. There was no valvular disease of the heart. There was pain over the heart region; he had bad teeth and suffered from indigestion; his pulse-rate was about 100. March 13, 1916: Faradic treatment and light physical training were begun; pulse-rate, 92. March 24: Often palpitation after physical training. April 5: Pulse-rate 80. April 13: Pulse-rate 76; is still on light physical training; general condition good; no palpitation for three weeks. April 25: Pulse-rate 76; will begin full physical training. After four or six weeks, as the man did not complain, and as the heart trouble did not get worse, he was promoted to the full physical training and the full route march, the duration of which is about the same as the lighter training, except that the exercises are more severe and the march longer and faster.

All the men were examined, very often during the exercises and also for some hours afterwards. Ten per cent. of the patients were unable to stand the light exercises at all at first. The following case is an example in point:—

Private H. was in France for eight months and was invalided in July, 1915; with palpitation, tachycardia and headache; pulse-rate 120. He had now and then palpitation before enlisting and during training. After a shell burst, he became worse and worse. Electrical treatment without physical training was commenced on March 2, 1916; pulse-rate 110. March 10: Pulse-rate 84. March 15: Pulse-rate 78. On March 17 light physical exercises were begun. On March 24: Pulse-rate 88. April 12: Pulse-rate, 100, uneasiness, palpitation; cannot stand the exercises; was excused all duties. April 26: Pulse-rate, 90, but feels better; will be recommended for discharge.

Fifty per cent. could stand the light exercises but not the full. The other 40 per cent. improved more or less quickly under daily treatment, and were soon able to stand the full course of gymnastics and marching. Those men who could not stand the exercises complained of excessive fatigue, of palpitation, of pain over the left chest, of breathlessness, and in most of them the pulse-rate was high, often 30 or 40 more beats persisting for one or more days afterwards. Some men who could stand the light exercises without any trouble were submitted again and again to the full exercises and each time the different symptoms returned. The following cases exemplify this condition:—

Case I.—Private G., aged 23, had had an enlarged thyroid for years; bad teeth, chronic throat catarrh, tachycardia (140 pulsations). Was at Gallipoli. The hot climate, very hard digging day and night made him weak. After a severe bomb attack he fell unconscious for an hour and experienced his first palpitation. On February 2, 1916, he began undergoing his treatment, light

exercises and faradization. After three weeks he felt much better, there was no palpitation, and the pulse-rate was 90. On March 2, he had full physical exercises, and on March 6, his pulse-rate was 120. On March 9, the pulse-rate was still as high as 115; and he complained of uneasiness, and some palpitation. On March 10, he was again on light exercises. March 17: Pulse-rate 88. March 23: Pulse-rate 72. On April 1 we tried him again on full physical training. On April 7 the pulse was over 100, and there was again uneasiness. On April 18 he was recommended for a Medical Board and was passed for Home Service, for which he was quite fitted.

Case II.—Lance-Corporal W., invalided May 15, 1915. He was in hospital for four months with crushed head. He was in France for three months and invalided for heart trouble. Before he enlisted he had an enlarged neck, but no heart or chest trouble. He first felt palpitations after two shell shocks, and was unconscious for a few hours. Stammering and tremor, good teeth, no constipation; pulse-rate 68. February 23, 1916: Electrical treatment and light physical training. March 15: Still light training; he has improved, his speech is much better; there are no palpitations. March 16: He began full physical training. March 22: Pulse still normal but complains of dizziness, headaches and palpitations. March 30: Pulse-rate 80; uneasiness. April 19: Pulse-rate 82; pain over heart region after physical training or full route march. April 26: Was on light physical training and light route marching; felt better; pulse-rate 72; was to be recommended for Home Service.

The electrical treatment consisted in daily faradization with a light current, of long wired coil, over the thyroid gland and the heart. This treatment is often employed on the Continent for exophthalmic goitre. It consists in an application with two tampons for three or four minutes over the thyroid, followed by three or four minutes with one tampon over the heart region, the other over the neck. This kind of current has a marked sedative action, which, however, is only local.

The static current may have as good and probably better effect, as it acts as a first-class tonic to the general nervous system, and local applications over the heart region may also be given by means of the static effluve or breeze.

Deep applications of the X-rays to the thyroid gland may also be very useful, especially so in the case of those men with hypertrophied thyroid (25 per cent. out of the total). Dr. Florence Stoney has described this treatment, and she obtains good results. The X-ray is certainly one of the best methods of treatment for exophthalmic goitre that we possess.

Tachycardia, excessive sweating, and nervous uneasiness are the symptoms which are the most quickly and completely removed.

The following are the results we have obtained after an average of two months' treatment: Out of those sixty men, fourteen were quite fit and returned to their unit; four others are fit and will return in a few days; others are on full physical training and will be fit in a short time; over a third, or 35 per cent., will be able to return to the firing line. Ten to 15 per cent. have to be discharged. The other 50 per cent. of the men have improved and may be fit for Home Service.

These first results are very encouraging and in the summer months I think we can do better. Outdoor life is easier; gardening, open-air games like bowls, quoits, rowing and swimming, may hasten the cure, while X-ray treatment, static electricity, and also hydrotherapy, will greatly augment our percentage of recoveries.

DISCUSSION.

Lieutenant-Colonel R. J. MORRIS, R.A.M.C. (Command Depot, North Camp, Ripon, and of Harrogate): Major McKenzie, in his admirable book entitled "Exercise in Education and Medicine," adopts the trite expression, "man is the sum of movements." How true this is! The value of physical exercises was recognized even before Greek and Roman times, but Professor Ling, rather more than a century ago, co-ordinated and elaborated physical exercises as a definite means of treatment suitable to many conditions. These remedial exercises, or medical gymnastics, may be divided into the (a) active, the patient doing the work himself; (b) the passive, all the work being done for him; (c) duplicated, in which the work is partly done by the patient, assisted, or resisted, by the operator. My experience in the Command Depot at Ripon satisfies me that the treatment by physical means has proved the most efficacious. Since November, 1915, just over 76 per cent. have returned to service in the various categories. The physical means adopted consist of massage, remedial gymnastics, without apparatus, conducted by experienced masseurs and masseuses, remedial exercises with apparatus such as pulley-weight machines, fixed-bicycle exercises, rowing machines, grip dumb-bells, graduated weights; also a most useful means of exercise and stimulation of nerves and muscles by means of the galvanic and faradic currents rhythmically interrupted and reversed, on the Bergonié system. This is a particularly useful means of exercise, because, in addition to the mechanical action upon the joints and muscles, we have the physiological trophic effect of impulses passing through the motor nerves. Further exercises are carried out by members of the Army gymnastic staff from Aldershot as soon as the convalescents are able to undergo and benefit by these more strenuous exercises up to the full physical course, under the supervision of a commissioned officer of the Army gymnastic service. The work requires much patience and perseverance,

because of the mental aspect of the convalescents, which does not help, and many cases are sent to the Depot much too late. Their disabilities are often "set," beyond the scope of exercises, which would not be the case were they treated by remedial physical means earlier. The Command Depot is an excellent institution, combining, as it does, drill, discipline, and treatment by physical, electrical, and other means; and even better results can be obtained, as the treatment at these depots becomes better known and understood by the authorities responsible for sending the cases. I welcome this discussion, and I feel that we owe Major McKenzie a debt of gratitude for his able paper.

Dr. W. BEZLY THORNE: I should like first to ask Major McKenzie whether, when these baths are given in cases of irritable heart and rheumatism, the water is in any degree mineralized, and if so to what extent.

Major TAIT MCKENZIE: The water is not mineralized at all, the effect depends on the temperature.

Dr. BEZLY THORNE: Perhaps I may be allowed to say, as one who has been engaged for more than twenty years in the balneological treatment of cardio-vascular affections, nervous and rheumatic affections, that I consider that this paper of Major McKenzie's marks a distinct epoch in the tardy and almost stagnant evolution of the therapy of cardiac diseases, vascular diseases, and allied affections. I only hope it may mark the beginning of a period in which, instead of the entire hopes of the profession being based on digitalis, strychnia, bromides, and strophanthus, they will look upon such things not as leading remedies at all, but as mere adjuvants to the physical treatment, which is infinitely more powerful, and which can produce a greater effect in four or five weeks than the other measures can bring about in as many years. I hope I shall not be considered presumptuous if I venture to suggest that in the treatment of those cases which are dealt with by baths, the water should be mineralized. I have had cases of trench rheumatism in which patients have been scarcely able to move, though they have been for weeks under treatment by radiant heat, massage, salicylates internally, and other similar orthodox measures; yet by subjecting these cases to the treatment adopted at many watering places of not drying the patient after the bath, but letting them dry in the blankets—and particularly if to the saline bath be added a certain proportion of what is now a common commercial article, radium salt—there has been an effect in a few weeks which sets these people entirely on their feet again. Not long ago I had a case in a young man, an officer, who had been through all these treatments, and was reluctant to undergo another, as he was sceptical about the result being good. I hardly like making this statement, lest I might appear to be exaggerating, but before this man had been long under the treatment—though when he came he could scarcely rise from the sitting posture—he was walking with full stride down to the War Office to ask for further employment. I therefore very respectfully suggest that in these establishments, which are doing such a wonderful work, such

admirable work, mineralization of the baths should be attempted. And another suggestion I venture to make is, that where there is an irritable heart and where there are tremors, it is very important not to allow the immersion to be too long. But mineralization of these baths is most effective, and I have not seen a single case since the commencement of the War in which both the rapid action of the heart and the tremors of the muscles and twitchings of the fingers have not ceased after the judicious application of such baths for five or six weeks, or in which the patient has not been well in two months. Therefore, with all respect, and with great admiration of everything I have heard this afternoon, I beg that those who are in charge of these valuable establishments will give attention to this point.

Dr. FORTESCUE FOX: As Major McKenzie has referred to the Committee of the Section of Balneology, I may be allowed, as in some sense representing that Committee, to make one or two observations. I think it is of very good augury that, probably for the first time in this country, a medical society has devoted an afternoon to the serious study of physical treatment in disease. And I was glad that it should have fallen to the Section of Surgery to make this departure, because I think that surgery and mechanical and physical treatment must always be, in the nature of things, very closely associated. I go all the way with Colonel Morris when opening this discussion, in believing that what has been now published will be useful as a guide to those who follow the same methods in other parts of the country; and I think that the reports which Colonel Morris and Major McKenzie have given us are most opportune.

With reference to what may be called the Hydrological Department at Heaton Park, I have two observations to make. I think that perhaps Major Tait McKenzie did not make it quite plain that the whirlpool baths are a new thing in our country, perhaps in any country, and that we are not, therefore, in a position to say anything very dogmatic about them, but from what we know at present we have reason to think that, given at high temperatures and over long periods of time, according to the individual requirement, they will materially assist in the movement of immobilized limbs. The experience which we are now gaining in London from day to day shows that they facilitate the use of massage and mechanical apparatus. Time alone will determine their ultimate value, and the place which they ought to occupy in a well appointed system of physical treatment.

With regard to the Pool Bath, I should like to felicitate Major McKenzie on being the first British subject who has had charge of a bath of that description. The flowing pool bath, at a constant temperature below blood heat, may be described as a powerful sedative to the nerve centres and circulation. This also is an entirely new departure in England, although not unknown in other parts of the world. Hereafter it will command more attention.

There are also two points in connexion with the use of physical remedies everywhere which appear to me to be important, and to which I trust anyone who may be in charge of physical treatments will give attention. The first is the necessity for accurate and periodical mensuration. In the London clinique we are working out this question, so as to determine not only the condition and degree of disablement at the beginning of the treatment, but the course of events from week to week, and what the exact final disablement may be. By means of apparatus, made partly in our own country and partly abroad, we are arriving, I hope, at a more satisfactory solution of the difficult question of mensuration.

The second of these points is with reference to records. I submit—and the Committee to which I have referred has repeatedly urged this matter upon those who may look to it for guidance—that it is very advisable that we should have, in all places where physical treatment is employed for wounded soldiers, a unified and uniform system of record. This recommendation has been made, not only in the interest of the patients, but also for the sake of medical science in the future, so that the opportunities afforded by this War may not be lost, and that in time to come we may know what measure of success has attended the use of the physical remedies that have been employed. I only wish, further, to add my congratulations to Major Tait McKenzie for the important piece of pioneer work which he has accomplished.

Lieutenant-Colonel WATT (Granville Canadian Special Hospital, Ramsgate): It has given me great pleasure this afternoon to listen to Major McKenzie's remarks, principally because we get the same class of cases at Ramsgate. Ours are cases of injury or disease of nerves, and diseases or injuries of joints, but we see them at an earlier stage than does Major Tait McKenzie. We find that, after all, as in every other branch of medicine, the diagnosis is practically everything. For this reason we have a Specialist Board, composed of Major Alwyn Smith, orthopædic surgeon, late assistant to Colonel Robert Jones, of Liverpool, and Major Colin Russel, neurologist, of McGill University, who, with their assistants, see every case which comes into hospital; and the value of their work, I take it, can be made out from the fact that fully 5 per cent. of the cases which go through our hands are psycho-genetic in origin. We are establishing a so-called "shrine" which is now a fairly large one, made up of sticks, crutches, jackets, splints, &c., of cases which are cured in from one to ten minutes, though they come in as instances of all kinds of paralysis, many of long standing. Captain Robertson, who is in charge of the laboratories, is working now on cases of myopathy and neurasthenia, and in six weeks he has discovered twenty-eight cases of tertiary syphilis among these patients, and under proper treatment there is no doubt they will soon be back in the firing line. In this, as I say, diagnosis is everything, for if a case is properly diagnosed, the treatment follows naturally, and succeeds. By our procedure, a case is either put under surgical treatment immediately, or is handed on for other appropriate treatment. Major Wilson is in charge of the therapeutics,

and he places each case under one or other of his assistants according to the nature of the disability. He has also under him a special medical officer who treats nerve lesions only, as we find that nurses and nursing sisters do not manage them properly. We have found that the results have justified this course. As soon as a man is able to do anything at all, he is given a chance of accomplishing some light work, and he is put into khaki with the regulation blue armlet to indicate that he is still a patient. If he abuses any privilege he immediately loses this. For this reason our "crime" is practically *nil*, because a man does not want to lose his khaki. If he does arts and crafts work he is transferred to an annex, to which is attached a large gymnasium. Here he comes under the medical officer in charge of gymnastics, who examines him and decides what special form of exercise treatment he ought to have. All our exercises are active: Major Smith is against passive exercises, except gentle manipulation during massage, preceded by *eau courante* baths. Most of the machines for giving the exercise we have made ourselves at our own workshop, and I think the total cost to the Canadian Government is only £100 for the whole equipment, which shows for how little money this can be done. A man may be put into an individual class, or a class for special groups of muscles, or finally into large classes of general gymnastics and Swedish drill. I have brought with me a table of what we do every day: Swedish drill and gymnasium, 30 per cent.; quadriceps class, 10 per cent.; tibial class, 15 per cent.; shoulder class, 5 per cent.; back class, 2 per cent.; elbow class, 8 per cent.; wrist class, 10 per cent.; special classes (flat feet, individual and artificial limbs), 20 per cent. As soon as a man is through with his exercises, under a capable instructor, he is passed on to another instructor, who puts him on to route marching, and as soon as that instructor is finished with him he passes him on to yet another, who supervises him on the playground, where he plays football, cricket, croquet, or anything else suitable to his disability: in this way he spends two hours every day in the open air in the fields. He also has to do light duty. He may have these duties in the hospital, or he may have to go to the arts and crafts department, which includes machine shops, printing, carpentry, cabinet making, fret sawing, cigarette making, painting and gardening. He is not permitted to work more than four hours a day, and may not work less than two hours. We have definitely to limit the time because some at least are so eager that they would try to go on for eight hours. In this way every man is employed at something the whole day long up to 4 p.m. We have had to increase their rations so much that many are now drawing the same full rations as the men in the trenches. In order to prevent over-fatigue or too prolonged stay in hospital, the checks on the men are four: (1) The medical officer examines every patient once a week; (2) we have them brought before the Specialist Board at least once in three weeks; (3) each patient must have his weight taken once a week and marked upon his own weight card; and (4) he must be seen once in three weeks by the officer-in-charge of therapeutics.

Dr. E. P. CUMBERBATCH (Electrical Department St. Bartholomew's Hospital): The treatment of war injuries by electrical methods, receives, it seems to me, but little attention in Major Tait McKenzie's interesting paper. He made no mention of static electrical treatment or diathermy or high frequency. Dr. Turrell will, I think, have some remarks to make on treatment by these electrical methods. My purpose is briefly to refer to the cases included in Major McKenzie's third class. Perhaps they could be included in a fourth class. I am referring to cases of paralysis following nerve injury, cases that recover very slowly and have been discharged from the Army. Some of these cases have come for treatment to my department at St. Bartholomew's Hospital, and it may be a year or longer before voluntary power is recovered and a useful limb saved. I think that such cases will derive most benefit, with the minimum expenditure of the time of the electrical department, from the rhythmically varying sinusoidal current applied in arm or foot baths. We have in the Electrical Department of St. Bartholomew's Hospital, a series of such baths, and numbers of cases of paralysis following injury or disease of nerve-trunks receive treatment there. We have obtained uniformly good results. Patience and the proper application of the current are the essential requirements.

Major W. J. TURRELL (Oxford): I beg to thank the Section for their kind invitation to me to attend and hear this interesting paper. It is most gratifying to hear this subject considered from the point of view of general physical therapy, which includes many sub-sections that are not often brought together. The paper this afternoon hardly, I think, exhausts the possibilities of modern electrotherapy in the treatment of the convalescent soldier. I am not quite clear what Major McKenzie means by the three-current treatment obtained from the multostat, or pantostat. These machines give the galvanic and sinusoidal currents; and although they are sometimes labelled as giving the faradic current it is the single phase sinusoidal and not the faradic current which is obtainable from them. There are two methods of electrical treatment which I think would prove exceedingly useful to Major McKenzie. First of all the diathermy current, a development from the better-known high-frequency current. The only action of this current is the production of heat. From the the most ancient times the value of heat as a therapeutic agent has been recognized, but all the former agents for its production, such as hot baths, hot air, radiant heat, ovens, &c., have only heated the surface of the body. The diathermy current can be passed through any part of the body, and it heats all the tissues through which it passes in its path between the electrodes. Any of the deep structures of the body can be heated in this way. Surely this is a great advance in thermotherapy. It has proved itself extremely useful in the relief of pain and is especially valuable in that form of severe and intractable pain which sometimes accompanies a traumatic neuritis. It has given excellent results in the treatment of trench feet. It is an absolutely safe current to use, provided that certain precautions are taken to prevent the

occurrence of burns. It affords an ideal current for the production of the relaxation of the tissues as a preliminary to massage and passive movement. Another form of electrical current, which we have found extremely useful at Oxford in the treatment of wounded soldiers, is that obtained from the static machine. There is no form of apparatus which has been more unjustly abused by the medical profession than this. A brief experience, however, with an efficient static machine would convince any unbiased medical man of its great utility, for it will accomplish results which can be obtained by no other form of electrical or other treatment. It is the apparatus which is in most request in our fully equipped electrical department at Oxford. By means of the very vigorous and almost painless contractions which it excites we obtain an entirely distinctive form of automuscular massage, which is capable of causing the absorption of synovial fluid from the knee-joint, of breaking down adhesions in the intramuscular fibres, and is capable of relieving the pain and limitation of movement occurring in old and extensive scars. The Bergonié apparatus for the production of electrically provoked exercises should prove of great value in these command depots. It excites a form of passive exercises which develops and trains wasted muscles to a very remarkable degree. By its use patients quickly regain the strength and tone of their muscles, and are then able by means of the active ergotherapy of their own exertions to complete the cure initiated by the passive ergotherapy of the electrically provoked exercises. The various forms of electrical treatment likely to prove of benefit to the convalescent soldier are far too numerous to discuss this afternoon, but I trust that the examples which I have given will show that the question of the further development and extension of electrotherapy should receive the serious consideration of those responsible for the treatment of convalescent soldiers.

Dr. GORDON (Exeter): There are one or two points on which I would like to say a few words. Major McKenzie will be thanked even by a larger audience than is gathered here this afternoon—an audience of men all over the country, who have been anxious to hear that the methods which he has been employing with so much energy and success are available for the wounded under their care. Command Depots similarly equipped are very nearly what many of us have desired to see established all over the country. One hopes that cases will in future be sent to them earlier; for there is no doubt, from what I have seen and heard, that it is absolutely deplorable how late many cases are sent for such treatment. Patients have been hurried from pillar to post, from one convalescent hospital to another, so that not uncommonly the period when there has been the most prospect of doing them good has long passed by. I trust everybody who is working in military hospitals will be informed that such treatment as has been described is at the disposal of their patients. A form of local heat which has not been referred to is that produced by means of hot sand. I regard it as a valuable form of treatment, and I have seen some cases benefit from it more than from radiant heat and light. You will

forgive me if I talk in units where others can speak in hundreds; but one case was that of a sergeant, with a knee apparently fixed in a horizontal position: his knee was buried in hot sand for twenty minutes every day, followed by gentle manipulation, and in a week or ten days his knee became flexible to a right angle. I am sure hot sand baking is a kind of treatment which should not be lost sight of. A question has been asked with regard to the value of medicated waters in the baths. At the request of some colleagues I went over to Paris last February, and the medical officers at the Grand Palais showed me everything that was possible. They took me to Engheilles-bains, a few miles north of Paris, where there is a sulphurated spa being turned to use for the treatment of injuries. They told me there that it did not seem to matter whether they used sulphurated water or plain water. But to answer the question on that point finally further observations seem essential. There has not been much said with regard to mensuration. The French have a system (the French are rather fond of systems, and I do not know that some of us are) and it has three clear advantages: First, every patient without exception goes through a certain series of treatments; secondly, the officers in charge soon become specially skilled in diagnosing the cases and arranging the treatments; and, thirdly, if one measures the disability at the start, and again at the end, a clear idea is obtained of just how much good is done, and it is easy to test the difference made by any modification of procedure. I think the lack of mensuration is a serious defect. With regard to the price of apparatus, we have seen and heard of many excellent suggestions for cheap forms of apparatus; but I cannot help thinking—and I hope Major McKenzie will forgive me for saying this—that, for instance, those which Dr. Fox has recently obtained from Paris and installed in London are better than the ingenious makeshifts which have been described. The Zander apparatus was very expensive because it was made of iron, but now twelve pieces, which do practically all that is wanted, can be bought for £175. With regard to the effect of heat and massage, we hear much of heat and much of massage separately, but few people keep the ideas together; yet it is essential that members of the profession should get it into their heads that heat and massage should be used together. There are masseuses who tell me that massage cases progress twice as fast when combined with radiant heat and light as without. If you use sometimes one, sometimes the other, separately, you will be making a mistake. I hope that those Government departments which deal with discharged soldiers will set their minds to work to try similarly to reduce their disabilities, which otherwise will be such a drawback in civil life when the War is over.

Dr. ACKERLEY (Llandrindod Wells): I have not had very many War cases, but for ten years I have treated many cases of the kind mentioned to-day by physical means, and I have practised them for twenty years. I treated a case of T-fracture of the elbow by massage in 1897. It is easy to classify these more or less chronic cases. A large number of these are

affections of muscles or joints: in these we have to cure immobility and restore functional activity. This is best done first by massage, then by passive, and last of all by active, movements. The reason for not beginning with active movements is that they give unnecessary pain. On this point I do not agree with the Canadian officer who spoke. As regards other cases, in a large group we have defective circulation or passive congestion of organ or tissue. Here to restore a healthy circulation we have recourse, temporarily, to an active *hyperæmia*. The means of producing this are many—and no dogmatic statement should be made as to the best means—but on the whole, dry heat is much preferable to moist heat; and radiant heat appears to me to act too much on the surface of the skin. It is to be deplored that English surgeons and doctors are not better acquainted with the work of Bier.¹ I should like to make a suggestion. I am living at one of the spas, and we have a large number of invalided soldiers whom we can only return as partially well. We have got rid of their pain and certain other disabilities, but we cannot get them into a condition in which they can march and carry a pack. Is it possible to make such arrangements as will enable us to send them to places where they can get Swedish exercises and marching? If Major McKenzie can make that suggestion to the War Office it will help us very much.

Major TAIT MCKENZIE (in reply): I have only a few words to say in answer to the points raised. The baths have not been mineralized because we have no facilities for doing so, and it is still a matter of dispute as to what good mineralization does. We depend entirely upon the temperature. I fear I did not make myself clear about the three currents. We have the faradic battery, giving the faradic current; we have the galvanic current, and we have the sinusoidal current. The high-frequency apparatus is expensive, and I believe it is still a matter of debate as to whether the expense warrants putting in an equipment of that kind in a Command Depot, but that is an administration question which it is better not to discuss. With regard to treatment by the Bergonié method, I think I am correct in saying that at the Command Depot at Ripon, under Colonel Morris, it is in use. I have not had personal experience of it, but I am sure that anyone who wishes can see it in operation at Ripon. With reference to the expense of apparatus, even £112 is to be considered if you can get the same effect produced by machines made by local people for a few pounds, but that, again, is a matter of administration.

¹ Bier, "Text-book of Hyperæmia," translated by Blick, 1909.

Surgical Section.

President—Surgeon-General Sir ANTHONY A. BOWLBY, K.C.M.G., A.M.S.

(July 12, 1916.)

(Chairman—Sir FREDERIC EVE, Lieutenant-Colonel R.A.M.C. (T.),
F.R.C.S.)

Cinematograph Demonstration of Methods of Bone-grafting.

By FREDERICK H. ALBEE, M.D. (New York).

THE series of cinematographs shown illustrated the growth of bone-grafts in the various surgical conditions in which they may profitably be employed, such, for instance, as Pott's disease, ununited fractures, and fractures of the neck of the femur. From the X-ray plates shown on the epidiascope, it was shown how rapidly these grafts become incorporated, gradually increase, and ultimately take on not only the function, but even the structure, of the bone they replace.

The main point in the technique of bone-grafting is that the graft must be kept as free from injury as possible. This is effected in the following manner: The groove in the broken bone which is to be filled up by the graft is cut by means of two parallel circular saws driven by electricity. These make parallel cuts in the broken bone, extending on each side of the fracture just as far as is required, and reaching down to the medullary cavity. A cross cut is then made at the end of each of the parallel cuts, by means of a small circular saw, and the piece of bone can then be easily lifted out. Similar saw-cuts are then made on another bone, the tibia by preference, so that a piece of bone

exactly the same size of the space prepared for it is obtained. This can then be placed in the groove cut in the broken bone; the surgeon can be sure of its fitting, and so quickly is the graft cut that it is not injured and its vitality is in no way impaired.

The various steps in the method are stated in fuller detail in the record of an illustrative case given below.

The second part of the Demonstration was an exhibition of cinematograph films showing the actual operations that have been carried out by the author.

Of recent years in the United States, a series of films have been taken, demonstrating surgical operations as performed by various experts. These films have been lent out on hire to the medical societies throughout the Union. The pictures have been taken at short range, and include only the operation field, to the exclusion of all unnecessary details. By means of the special lighting arrangements adopted, the obscuring effect of the blood is seen to be reduced to a minimum, and every detail, from the first incision to the application of the final dressing, can be clearly followed.

A film was shown illustrating the treatment of a case of ununited fracture of the tibia. The case had been treated by the application of a metal plate, without success; sinuses formed and the presence of a definite sequestrum was demonstrated by the X-rays. The case was subsequently treated in two stages (both shown on the film): the metal plate and sequestrum were first removed and the wound plugged. After an interval, the wound having meanwhile firmly healed, without, however, any union in the tibia taking place, the seat of fracture was exposed by turning out a flap, and the fibrous tissue removed between the fragments of the bone. The next step in the operation, consisted in the removal of a strip of bone about $\frac{1}{4}$ in. in breadth, and about 5 in. long, from the two fragments, that is to say a gutter had been cut in each, extending upwards and downwards on either side of the seat of fracture for a distance of about $2\frac{1}{2}$ in. This was carried out with the author's electrically driven double saw,¹ which consists of two circular saws set on a central axis, so that a long strip of bone of the same width throughout its entire length is removed. A stream of saline is allowed to flow on the saws to prevent any overheating. In this way a bed was formed for what is styled an inlay graft. The graft was next cut with the

¹ This is illustrated in the paper by Lieutenant-Colonel Robert Jones, "Transplantation of Bone and some uses of the Bone-graft," *Brit. Med. Journ.*, 1916, ii, p. 3.

same saws from the other tibia, so that a strip of bone was procured consisting of the four elements of bone, and accurately fitting the bed that had already been prepared for it. The graft was fixed in position by strong kangaroo tendon sutures, which passed through holes drilled by the motor on either side of the gutter. The tendons were threaded through from one side of the gutter to the other, then pulled up from the gutter in the form of loops under which the inlay graft was forced into place, and the tendons then tied over it.

The value of bone-grafting is especially indicated in dealing with those fractures in which there has been great destruction of bony tissue, leaving an inch or more separation between the fragments.

There have been cases in which a successful result has been obtained, even though the graft spanned an area where active tuberculous disease was present, the essential being that the graft should have a good contact with healthy bone at either extremity.

The main feature of the electrical apparatus, consists in the tools being driven by a motor in a sterilizable casing in the operator's hand.

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The Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.

Therapeutical and Pharmacological Section.

President—Dr. W. HALE WHITE.

(October 19, 1915.)

DISCUSSION ON THE TREATMENT OF CEREBRO-SPINAL MENINGITIS.

Opened by Sir WILLIAM OSLER, Bt., M.D., F.R.S.

FOR the first time in the medical history of these islands cerebro-spinal fever has prevailed as an epidemic, and with the usual characteristics—a low case-incidence, a widespread but capricious distribution, a predilection for soldiers and for young soldiers, a high case-mortality, and a rapid disappearance of the disease with the approach of fine weather. The figures of its distribution and prevalence are not yet available. As is usual in Europe, the military population has suffered most. In the first year of the War there were, in the Home Army, 462 deaths, which put cerebrospinal fever second on the list among the causes of death from acute infections, probably a very much larger number than among the civil population. When the returns are out we shall find that the deaths from cerebrospinal fever form but a small proportion of the total deaths from meningitis. It is to be hoped that one result of the recent careful studies will be a more accurate registration of the forms of the disease. For 1913 the Registrar-General reports 9,269 deaths from meningitis in England and Wales. More than half were the tuberculous form, 5,018; of the balance, 159 were classified as posterior basic, 163 as cerebrospinal fever, and 3,819 as "other forms." We need information as to that last group, in which there may be a not inconsiderable number due to the meningococcus. Not only as an epidemic has cerebrospinal fever

interesting peculiarities, but as a meningitis it has the unique and fortunate distinction of being the only variety from which recovery takes place in from 50 to 70 per cent. of the cases. Many men of long and rich clinical experience have never seen recovery in the tuberculous, pneumococcic, or streptococcic varieties. For the first time I saw this winter a case of the latter get well, the bacteriology of which was worked out by Dr. Gordon.

That cerebrospinal fever is a specific infection due to a special germ is universally acknowledged. The meningococcus is always present, it may be grown in pure culture, it has specific serological reactions, and the disease may be reproduced in animals. A hard organism to deal with, it has cracked the reputation of many "half-baked" bacteriologists. Like Eberth's bacillus and the pneumococcus, it has "strains" not yet worked out satisfactorily, and the existence of which has not been sufficiently considered, as they present differences in agglutination, opsonization, and complement-fixation.

Only two points call urgently for discussion—the prophylactic measures and the serum treatment; others are, in comparison, subsidiary and unimportant.

The history of the epidemic may lead us to expect a return of the disease in the winter months. There are instances, indeed, of a progressive yearly increase in the severity of the outbreaks. What preparation shall we make? In the first place, let us recognize a certain value in the new conception of the disease propounded by Dopter, and supported in this country by Arkwright, Lundie, Thomas, Fleming, and MacLagan. The epidemic is in the carrier, the meningitis is an incident. The germ is ubiquitous, harboured by many who are not ill and who never have any symptoms, producing in others a mild catarrh, and only a few reaching the meninges. After all, this is just what happens in pneumonia, in which the proportion of cases to carriers is even smaller than in cerebrospinal fever. We cannot recognize always a catarrhal or nasopharyngeal stage of the disease, and the germs may be present in a healthy-looking throat of a serious case. Two broad facts stand out—the correlation of the seasonal prevalence with nasopharyngeal catarrh, and the influence of overcrowding in ill-ventilated barracks and houses. The concentration of human beings, particularly of young recruits, in camps appears to be the most important single factor. Fatigue lowers the resistance, wet and cold and excess of tobacco favour pharyngeal catarrh; too often the huts or tents are overcrowded, the nights are cold, the blankets thin—what

wonder if the men close every window, or lace up the tents tightly, and that the air becomes foul? A carrier does the rest—distributes the germ to some young fellow whose resistance is weakened or whose nasopharynx forms a suitable medium.

During next winter the medical officers in charge of camps should (1) guard the young soldier specially against over-fatigue; (2) reduce to a minimum the circumstances which favour nasopharyngeal catarrh; and (3) make a combination possible of good ventilation of the sleeping quarters with comfortable warmth of the men. These may not be easy details to carry out, but they appear to be essential; and they are worth fighting for to save, possibly, the lives of 500 young men. In the *Lancet* of this week¹ Dr. Halliday Sutherland has offered important suggestions of the same character, and I do hope those in charge of the camps will use every effort to prevent a recurrence of the outbreak.

The prophylactic use of the anti-meningitic serum is still on trial, and those who have had experience will doubtless forward their results.

With our present knowledge a specific therapy combined with lumbar puncture is the rational treatment, combating with the one the sepsis and with the other the pressure effects of the local exudates. We are all agreed upon the value of withdrawal of fluid from the spinal meninges, the technique and various details relating to which scarcely need to be discussed. On the value of the specific therapy there is grave difference of opinion.

The use of serum is based on sound experimental data. The long and elaborate studies of Jochmann and of Flexner and his pupils have made possible the production of a serum which has a specific influence on the meningococci in the spinal canal, partly by a direct bactericidal action, partly by promoting phagocytosis; possibly, too, there is an anti-endotoxic action. I have followed with the keenest interest the Rockefeller work in this subject, and it is sound and scientific, a model of accurate and prolonged research.

The specific treatment has been in use now for some years, and details of the results are to be found in the recent monograph of Sophian. Table XXVIII gives the collected statistics of cases treated with and without the serum; and if worth anything—which in these iatro-mathematical days many doubt—the figures show an overwhelmingly strong case for specific therapy. In view of the possible repetition of the outbreak next winter, the practical points for us to discuss are

¹ *Lancet*, 1915, II, p. 862.

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¹ *Lancet*, 1915, ii, p. 862.

(1) the experience of the past few months, and (2) its lessons for the future.

I am afraid we must confess to disappointment in many quarters with the serum treatment. The reasons for the failure should be laid open freely and frankly. What we wish is to get at the truth of the matter. My personal contribution is not worth much. A consultant sees only the severe cases, and usually when far advanced. A general impression remains of a type of epidemic unusually severe, and certainly the death-rate has been high. I hope the figures for the whole country may soon be available.

I was much interested in the outbreak among the Canadians at Salisbury, and have to thank the medical officers, particularly Colonel Murray McLaren, for opportunities of seeing the cases. I have asked Captain Ellis, who was in charge of the treatment at Bulford, to send me a report, and this he has done through the kindness of Surgeon-General Carlton Jones, of the Canadian Forces. Dr. Ellis has been for several years in charge of the special work on tabes and general paralysis at the Rockefeller Hospital, New York, and is an expert in the technique of spinal therapy. There were forty cases, with twenty-six deaths, a mortality of 63 per cent. The sera used were from Burroughs Wellcome and Co., the Lister Institute, Mulford, and Parke, Davis and Co. "A few of the patients treated with Mulford's serum suggested that some improvement had taken place, but this was hardly striking enough to justify its use. No benefit was seen with any of the other sera employed. In one patient meningococci were isolated from the spinal fluid two and a half months after the onset of the illness. During this period the patient had been given more than twenty intraspinal injections, comprising all of the above kinds of serum. In another case meningococci were in the fluid five weeks after the onset of the illness, during which time the patient had twelve injections of serum." This is a bad record for the treatment, and all the worse coming from the hands of an expert in intraspinal methods. Nor were the results any better at Haslar or Shorncliffe. Staff-Surgeon Robson and Captain A. L. Pearce Gould have reported a group of thirty-one cases, some of which I had the opportunity of seeing with them and with Surgeon-General H. D. Rolleston. The mortality was 52 per cent., and the use of the serum was abandoned after the occurrence of eight deaths among twelve cases.

In other local outbreaks there is the same unfortunate story, but happily, I believe the man of largest experience, Dr. Robb, of Belfast,

will be able to tell another tale. We want to know in this discussion the reasons for this widespread failure. I think there has been one main cause—inert sera. The strain or strains causing the meningitis were “fast” to the sera employed. We do not know in how many cases the para-meningococcus was present, and it possesses specific agglutinins and precipitins and does not react to the ordinary anti-meningitic serum. Dr. Gordon and others will be able to enlighten us on those points. Insufficient dosage, failure to treat cases early, imperfect technique, have, I believe, been minor contributory causes to the failure. Of course, there are cases that resist all treatment, and there are complications which together will always keep the mortality at $\frac{1}{4}$ per cent.

We are face to face with two problems, the only ones worth discussing: How we may head off another outbreak next winter; but should we fail in this, how may we get our mortality reduced to 25 per cent. by profiting from the hard lessons of the past few months?

This should be made a War Office problem; it should be put into the hands of experts, who can investigate sera, so that we could be certain they corresponded to the strains of organisms present in the prevailing epidemic.

Dr. GARDNER ROBB (Belfast).

He had always been greatly interested in the subject, as they in Belfast and the North of Ireland had suffered severely from outbreaks of this disease since 1907.

Sir William Osler had suggested that the discussion that day should run under two heads: First, prophylaxis, and, secondly, the treatment of the disease. As prophylaxis was not his province, he would confine his remarks to treatment.

Before the introduction of the serum of Flexner there occurred in Belfast a severe outbreak. In the first seven months of that outbreak he had under his care in the fever hospitals 275 cases, and the death-rate was over 72 per cent. Then he received from Dr. Flexner a supply of the serum, on the preparation of which he had been working for a long time. It had then been tried in a few scattered cases in America with good results. He began its use in the beginning of September, 1907. The change in results was dramatic. The death-rate fell at once to one-third of what it had been. The death-rate in his hospital

cases had been over 80 per cent. in the three months immediately *before* the serum arrived. Of forty-five cases admitted in those three months thirty-seven had ended fatally. In the first four months *after* the use of the serum was begun the mortality in hospital was 26 per cent. Only half the cases occurring in the city at that time were sent into hospital, and during the same four months the mortality in the cases not sent into hospital and not treated with serum was 85 per cent. Since then he had frequently reported the result he had obtained with Flexner's serum, and in no series of cases up to the beginning of last winter's epidemic did the mortality in his Belfast cases so treated exceed 30 per cent.

No one who had had any considerable experience in the treatment of that disease without intraspinal injections of serum and then had an opportunity of observing the results of such injections could have any doubt as to the enormous value of that treatment; and he believed he was correct in stating that *everyone* who had had such opportunities was enthusiastic regarding the success of the method. To that he thought there was not any exception, as shown by the writings of Dunn, Emmett Holt, Ladd, Royer, Sophian, Fulton and many others in America, of Netter and Dopter in France, of Claude Kerr, of Edinburgh, and of Christomanos with his large experience in Greece. The same unanimity was to be found in the records from Germany, where similar success was obtained after the adoption of the intraspinal route for the administration of the antiserum.

In the discussion at the Epidemiological Section held last February he had reported his results to that date. The number of cases was then about 120, and the mortality had been under 24 per cent. Those were gross mortality results; *all* the deaths were included, and he would again plead that gross mortality-rates should be given in records of serum-treated cases for two reasons: first, because the death-rates of 60 to 80 per cent. commonly reported in outbreaks in America and Europe before the introduction of intraspinal injections of serum were gross mortality-rates; and, secondly, because from his experience he believed it was quite impossible to say in the first few hours of illness that any case was hopeless.

In a recent and very excellent work on cerebrospinal meningitis the author, who had large experience of the serum treatment in a comparatively recent epidemic, and had a gross mortality-rate of 25 per cent., had excluded over 40 per cent. of his fatal cases because they were "hopeless from the first," or died within twenty-four hours, &c.

He then published a table of cases reported from various places all over the world. The mortality from these places was a gross rate, whereas the author's was a "corrected" rate. Naturally his results were about twice as good as those obtained by others.

To illustrate how impossible it was to give a definite prognosis, he might be allowed to mention one case which occurred last February. He was asked late one night to see a young man who had only been taken ill that morning. He had been in excellent spirits on going to bed the night before, was unable to rise in the morning on account of severe headache, he soon became delirious and then unconscious, and when Dr. Robb saw him some fifteen or sixteen hours after the onset he was unconscious; the pulse could not be counted at the wrist, there was great rigidity, and he was covered with large patches of subcutaneous hæmorrhages and a plentiful petechial eruption; on his feet there were large hæmorrhages into the skin measuring 2 in. by 3 in.; his ears, hands and knees were covered with deep purple patches of hæmorrhage, and there were large subconjunctival hæmorrhages. Though it seemed doubtful to Dr. Robb and to his own medical attendant whether he would survive the four or five miles' journey to the hospital, it was his only chance, and they sent him in. He was punctured immediately after his admission; the cerebrospinal fluid was purulent, and meningococci were plentiful, both intra- and extracellular. He was given 40 c.c. of serum. Twelve hours later he was given 20 c.c., but seemed no better, and the whole cornea in one eye was steamy with pus in the anterior chamber. He was given injections daily for several days and slowly improved. Eventually he recovered with the loss of one eye; some patches of skin became necrosed and sloughed. He left hospital after ten weeks' detention, and lately Dr. Robb had heard from his doctor that the patient now reported himself in better health than ever before, and he was earning his living as a carpenter on munitions work. This case was quoted to show how impossible it was to separate cases which "had no chance" from other severe cases. If this patient had died he might readily have been put in the category of cases which were "hopeless from the first."

They had remained free from the disease in Belfast in 1913, and to the end of 1914. When there was a reappearance of cases last winter he had fully expected that they might look forward to getting even better results, as owing to the experience they had had the diagnosis was more likely to be quickly made, and the value of early injection of serum being fully recognized, the cases were more likely to

be sent into hospital without delay. It was also apparent that they would have many cases from the camps, the patients in such cases being generally in good physical condition, and at a time of life when good results were to be expected. But unfortunately the results, instead of being better, had not been so good. Since the beginning of last winter he had had ninety-two cases in hospital and had treated eight cases in various places outside, making exactly 100 cases; of these thirty-six had died, a case-mortality of 36 per cent. In the total number they had had previously the death-rate had been 24 per cent.

In trying to find an explanation of the cause of this increase, several questions occurred to one:—

- (1) Was the disease of a more virulent type?
- (2) Was there any marked difference in the average age of the patients which might make for a higher mortality?
- (3) Could the increase be accounted for by differences in the strains of the meningococcus?
- (4) Was the serum available of as high standard of immunity value?

(1) Regarding the virulence of the type, it was, of course, very difficult to make a correct estimate, but his opinion was that the recent outbreak had not been more severe in type than formerly, although so far as he had been able to ascertain, no case occurring in the North of Ireland during the past winter and spring which was treated without serum had recovered. There had been several such. Unfortunately, the disease was not universally notifiable in the various sanitary districts, with them, so that it was not possible to obtain exact figures.

(2) The average age of the patients had been higher in the recent outbreak. This should, on the results generally reported in former outbreaks, make rather for a lower case-mortality, but his own experience had been that young children gave very good results when treated with serum, provided they came under treatment reasonably early. It was in the infants that the greatest improvement in results had been obtained since the serum treatment became general. In children under 1 year treated without serum a death-rate of 90 per cent. was common. With the serum they did quite as well as cases in other age-periods. In the present outbreak he had had seventeen cases in children under 5 years with only four deaths—less than 25 per cent. (One of these fatal cases was already over fifty days ill when admitted from a country district twenty-five miles from the city of Belfast.)

- (3) Was the increase in the mortality to be accounted for by

differences in the strain—strains not provided for in the polyvalent serum available? In the discussion at the Epidemiological Section last February he believed that the general impression held by many of those taking part was that the resistance to the serum treatment shown by many of the cases then occurring was to be thus accounted for. During the early spring he had had several cases which were received into hospital quite early in the attack, but though living long enough to receive several daily injections, had shown no improvement or only very temporary improvement and ended fatally. He was aware that cases of that kind had been met with amongst the serum-treated cases elsewhere; personally he had not met with such cases in his former experience in the use of the serum. His experience had been that if a patient lived for ten days or a fortnight the case recovered. In this outbreak that did not hold good. He might instance one case which occurred amongst those recently under his care: a robust soldier, aged 20, was admitted with a severe attack on the first day of illness; he received repeated injections, showed some improvement at first, but only for a short time. In all he was given fifteen injections—480 c.c. of serum—and had over 1,100 c.c. of fluid drained off from the canal. He went steadily downhill and died after four weeks. The meningococcus was present in the cerebrospinal fluid till the end. In his former series he had not met with such cases. Having had some correspondence with Dr. Flexner on these resistant cases, he had collected specimen cultures of the organism from cases in various places and had taken them over to New York for comparison with the strains being used in the preparation of the serum. Through the kindness of Dr. Martin he had obtained several from the Lister Institute; from Dr. Robertson he had received some from Birmingham; he had also received cultures from Professor Beatty's laboratory in Liverpool, and from Professor McWeeney, of Dublin. Most of these he had tried to take out in sealed tubes, but these all died off on the voyage; but he had taken a small incubator and had transplanted samples from all these places on the voyage, so that strains from the various districts were available for comparison. These had shown no marked differences when compared with those in use in the preparation of the serum now being made at the Rockefeller Institute. Some of them had not agglutinated with the serum in quite so high dilutions as those in use there, and these have been used in the preparation of the serum now. Dr. Amoss, who was working with Dr. Flexner at the Rockefeller Institute, had promised to send him a fuller report, but he had not yet received it. With the

recent improvements in culture media, interchanges of cultures for comparison should be less difficult.

(4) Was the serum available of as high a standard of immunity value? For two or three years before their epidemic last winter there had been very little demand for the serum from the sources of supply in America; then suddenly came the great demand from this country. Our War Office ordered large quantities, and much was requisitioned from France, with the result that the available horses were bled as frequently as possible, and the immunity value of the serum dropped very seriously. For this and possibly for other reasons he thought there was no doubt that much of the serum which had reached this country last winter was of a much lower standard of value than that formerly supplied. He thought that therein lay the true reason of the somewhat disappointing results sometimes obtained from its use. Steps had been taken to correct the deficiency in the quantity and the quality of the serum. Dr. Flexner had also again taken up the preparation of serum at the Rockefeller Institute, and serum was now available which, judged by all the laboratory tests, was of higher value than any formerly in use. He had brought back with him a supply of this new serum, and so far he had treated eight acute cases with it; in these there had been one death. This was very promising, but was, of course, much too small a number to judge by. Some of these eight cases were comparatively mild, but some were quite severe types. The latest case, a soldier, admitted nine days ago—then twenty-four hours ill—was deeply unconscious, with normal temperature, cyanosis, stertorous breathing, and the dreaded slow pulse. On puncture thick syrupy pus was obtained. He was given 40 c.c. of serum; next day he could be roused, but had paralysis of one side of the face. The following day there was paralysis of the arm on the same side. He then steadily improved, and when Dr. Robb saw him the previous day he was quite bright, the paralysis of the face had cleared up and the arm nearly so, and the meningococcus had disappeared from the fluid. He appeared to be on the fair way to rapid recovery.

He hoped that some of the speakers that day would give them their views on the advisability of taking continuous blood-pressure readings during lumbar puncture and the injection of the serum, as insisted upon by Sophian, also on the use of general anæsthetics. He confessed that when he first read Sophian's note on the value of blood-pressure readings and on the danger of general anæsthetics he felt uneasy. He had not at that time—some three years ago—had any cases for a considerable

period, but he felt that possibly the cases he had treated had not the best of chances as he had never been guided by blood-pressure readings, either in the quantity of fluid drained off or in the dosage, and he had generally given chloroform. Early in his experience he received a lesson which he had not forgotten. He did not like giving chloroform to these patients so frequently, though the patients so often asked for it. In one of the first cases he ever had—a powerfully built man—he had given chloroform at the first injection, and when repeating the injection he explained to him what he was about to do and he consented to bear it without moving if possible. The patient bore the puncture with quite a large trocar without complaint; the fluid came freely and all was going well, when suddenly, Dr. Robb supposed through some slight movement of the trocar, the patient felt what was evidently excruciating pain; he sprang off his side and made desperate efforts to bury his back in the bed so that Dr. Robb would not again be able to touch the trocar. The patient was immensely muscular; Dr. Robb had one assistant and a nurse, but he was very greatly relieved when he had at length succeeded in getting the trocar removed. He completed the injection under chloroform. Since then he had always used a general anæsthetic unless the patient was unconscious or in a few cases where there seemed to be some contra-indication. From Dr. Robb's experience of some 800 or 1,000 injections, quite 80 per cent. of which had been done under general anæsthesia, he believed these patients gave no more anxiety to the anæsthetist than any others, and he would recommend the use of a general anæsthetic unless where obviously unnecessary or for some reason contra-indicated. He had not been able to obtain success with "water anæsthesia" as recommended by Sophian. In some cases he had tried taking continuous blood-pressure readings, but did not think he received any help from them. With general anæsthesia the variations in pressure did not seem to be marked either during the withdrawal of fluid or the injection of the serum. The withdrawal of fluid often gave rise to very severe pain, and, as Sophian said, the injection at times gave rise to pain which might be almost unbearable. Dr. Robb thought the explanation of many of the alarming variations in blood-pressure was to be found in this sudden severe pain set up. With general anæsthesia this was avoided. As the result of his experience in this method of control of dosage Sophian had found that much smaller doses of serum appeared to be the safer course, and recommended that in young children the dose should be 3 c.c. to 12 c.c. In Dr. Robb's cases in young children he had always given the full dose of 20 c.c., often

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more, generally under complete anæsthesia, and the results had been quite good. In the series he had reported that day were included two children under 1 year—both had recovered. These were not mild cases. One, aged 6 months, admitted on the fifth day, required seven injections, the last on the nineteenth day; this case had received in all 150 c.c. of serum—viz., six injections of 20 c.c. and one of 30 c.c.

The best dosage for any age, infant or adult, had not, of course, been as yet worked out; personally he was inclined to give full doses and frequently repeat them until definite improvement was shown. He was quite sure they often used more serum than was necessary, but at present they had no means of estimating the risk of withholding it in cases not distinctly improving.

Though he must confess to some disappointment with the results in this series of cases, and with the mortality of 36 per cent., he thought there could be no doubt that, should there be a recrudescence of the disease in the coming winter, they might look forward to having available serum of much higher value than much of that which reached this country from America last winter.

Surgeon-General H. D. ROLLESTON, R.N.

From the commencement of the War to July 31, 1915, there had been about 170 cases of cerebrospinal fever in the Royal Navy.¹ The following summary of the results of treatment was abstracted from the notes of 163 cases, 89, or 54·6 per cent., of which proved fatal. The prolonged and unsatisfactory cases naturally received more varied treatment than the fulminating cases, some of which died shortly after coming into hospital. As would be seen by the tabular statement, various forms and combinations of treatment were employed, and the most noticeable point was the failure of intrathecal injection of antimeningococcic serum.

Antimeningococcic serum from various sources (Burroughs Wellcome and Co., the Lister Institute, and Mulford) was employed.² In 105 cases the treatment consisted of lumbar puncture and intrathecal injection of the serum, either alone (62 cases) or with the addition of vaccines, soamin,

¹ Being an extract from a report made to the Medical Director-General, and to be published in the forthcoming number of the *Journal of the Royal Naval Medical Service*.

² In one case, at Deal, 10 c.c. of blood serum from a patient convalescent from the disease was injected intrathecally the day before death.

or hexamine (43 cases). Of these 105 cases 64, or 61 per cent., died; and 41, or 39 per cent., recovered. Of the 62 cases treated by lumbar puncture and intrathecal injection of serum death occurred in 43, or 69·4 per cent., and recovery in 19, or 30·6 per cent.; whereas of 43 cases treated in addition by vaccines, soamin, or hexamine, 20, or 46·5 per cent., were fatal, and 23, or 53·5 per cent., recovered. It was therefore

	Cases	Deaths	Recoveries
<i>Antimeningococcic serum—</i>		Per cent.	Per cent.
Intrathecally	105	64 or 61·0	41 or 39·0
Alone	62	43 or 69·4	19 or 30·6
Combined with vaccines, soamin, hexamine, or serum hypodermically	43	20 or 46·5	23 or 53·5
Combined with soamin	18	11 or 61·0	7 or 39·0
Combined with an auto-vaccine	11	2 or 18·2	9 or 81·8
Combined with hexamine	7	5 or 71·4	2 or 28·6
Combined with serum hypodermically	7	2 or 28·6	5 or 71·4
Hypodermically	19	6 or 31·6	13 or 68·4
Alone	4	1	3
Combined with intrathecal injection of serum	7	2	5
Combined with auto-vaccine and intrathecal injection of serum	4	1	3
Combined with an auto-vaccine	3	2	1
Combined with soamin	1	0	1
<i>Autogenous vaccine (never alone)</i>	16	4 or 25·0	12 or 75·0
Combined with serum intrathecally	6	1	5
Combined with serum intrathecally and hypodermically	4	1	3
Combined with serum intrathecally and soamin	1	0	1
Combined with serum hypodermically	4	2	2
Combined with soamin	1	0	1
<i>Soamin</i>	43	19 or 44·0	24 or 56·0
Alone	21	7 or 33·3	14 or 66·7
Combined with serum intrathecally	18	11 or 61·0	7 or 39·0
Combined with serum intrathecally and hexamine	1	1	0
Combined with serum intrathecally and vaccine	1	1	0
Combined with serum hypodermically	1	0	1
Combined with an auto-vaccine	1	0	1
<i>Lumbar puncture (alone)</i>	13	4 or 30·8	9 or 69·2
<i>Symptomatic treatment (only)</i>	14	10 or 71·4	4 or 28·6

obvious that the cases treated solely by the intrathecal injection of serum and lumbar puncture had a higher death-rate than that (54·6 per cent.) of the whole series of 163 cases.¹ Flexner had laid much stress on the importance of injecting antimeningococcic serum intrathecally

¹ Flexner, *Journ. of Exper. Med.*, New York, 1913, xvii, p. 553.

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as early as possible in the disease; and in a table dealing with 1,211 cases he had shown that when the serum was injected within the first three days the mortality-rate was 18 per cent., when between the fourth and seventh days 27.2 per cent., and when later than the seventh day 36.5 per cent. The following tabulation of 105 cases treated in the Royal Navy had been made on the same lines for comparison:—

	Cases	Deaths	Recoveries
First to third day ...	70 ...	42 or 60.0 per cent. ...	28 or 40.0 per cent.
Fourth to seventh day...	24 ...	14 or 58.4 " ... 10 or 41.6 "	
Later than seventh day	11 ...	8 or 72.7 " ... 3 or 27.3 "	

The failure of antimeningococcic serum to reduce the mortality was therefore not due to its being given too late, for in 66.7 per cent. of the 105 cases it was administered within the first three days of the disease. The serum treatment, which was so successful in America (Flexner, Sophian), Belfast (Robb), and elsewhere, was given a thorough trial and proved most disappointing. In a very few instances was there the critical improvement which was said to occur in about 30 per cent. of the cases that recovered (Flexner).¹ Possibly the meningococci were "fast" to the action of the serum or were para-meningococci. In consequence of its apparent inertness intrathecal injection of serum was in the latter part of the epidemic largely replaced by or combined with other methods, such as the intramuscular injection of soamin. It was true that the mortality naturally diminished towards the end of epidemics; but this did not entirely explain why, as shown by the tabular statement, the results of intrathecal injection of serum compared so badly with those of almost all the other methods and combinations of methods; for up to the end of February there were 90 cases with a mortality of 52, or 57 per cent., whereas from the beginning of March to the end of July there were 80 cases with a mortality of 38, or 47 per cent. There was, for example, a remarkable contrast between the effects of intramuscular injections of soamin: (a) when given alone to 21 cases, with a mortality of 33.3 per cent., and (b) when combined with intrathecal injection of serum in 18 cases, with a mortality of 61 per cent. It must be recognized that when dealing with small numbers fallacies easily crept in, but merely from these figures the addition of intrathecal injection of serum would appear to have coincided with an increase of mortality in the cases treated with soamin. The high mortality could hardly be

¹ Quoted by Heiman and Feldstein, "Meningococcus Meningitis," 1913, p. 282.

explained by the suggestion that it was due to the bad effects of lumbar puncture, for in 13 cases in which lumbar puncture alone was employed there were 9, or 69 per cent., recoveries; and in 91 per cent. of the 163 cases lumbar puncture was performed, and in many cases repeatedly, to relieve symptoms referable to increased intrathecal pressure. Alarming symptoms directly after the intrathecal injection occurred in two cases only, thus showing that the gravity method of administration, which was not in vogue in the Royal Navy, was hardly necessary provided due care were taken. Serum rashes were noted in 20 per cent. of the cases, but were probably more frequent; in a few cases there was also arthritic pain. No severe anaphylactic symptoms were recorded.

Antimeningococcic serum was given hypodermically in 19 cases; but, as in four cases only was it the sole form of specific treatment employed, no conclusions as to its influence could be drawn.

An autogenous vaccine was given in 16 cases, mainly by Fleet-Surgeon H. C. Whiteside, R.N., at Plymouth, where the mortality (36.7 per cent.) was remarkably low. The cases to which auto-vaccines were given all received other specific remedies (in 11 cases intrathecal injections of serum), and showed the very low mortality of 25 per cent. But as the numbers were small this result—though a further stimulus to its more extended use—must not be insisted on.

As already mentioned, soamin appeared to give good results; its beneficial effect in the septicæmic stage of the disease was noticed by Staff-Surgeon B. S. Robson, R.N., at Haslar, and had also been mentioned by others.¹ In one case at Portland Hospital as much as 42 gr. of soamin were given. Optic atrophy was not noted in any instance.

Hexamine was given by the mouth in seven cases in the hope that, as it was secreted into the cerebrospinal fluid, it would exert a bactericidal action on the meningococci; but as it did not appear to have any effect clinically, it was soon abandoned.

Lumbar puncture, which was such an important means of diagnosis, was performed in 149 out of 163 cases, or in 91.4 per cent. In 38 cases lumbar puncture was done once only, but 14 of these cases died soon after they came under observation; six other cases, tapped once, died. Of 22 cases, tapped twice, 8 proved fatal, 2 being very acute; 3 tapplings were done in 20 cases (9 deaths), 4 in 24 (15 deaths), 5 in 15 (9 deaths), 6 in 6 (3 deaths), 7 in 7 (4 deaths), 8 in 3 (2 deaths), 9 in 4 (all fatal), 11 in 5 (2 deaths), 13 in 1 (fatal), 15 in 1 (fatal), 16 in 1 (fatal), and 17

¹ Batten, *Lancet*, 1915, i, p. 966.

in 2 (both recoveries). Lumbar puncture appears to be a palliative rather than a curative remedy and to relieve for a time symptoms due to increased intrathecal pressure. In 13 cases, of which four proved fatal, it was the only form of treatment other than the ordinary symptomatic remedies employed.

In 14 cases, 10 of which proved fatal, symptomatic remedies only (such as morphine for pain) were employed.

With regard to prophylactic measures, it seemed advisable in the case of the Services to invalid the men who recovered from an attack of the disease, because they might be intermittent or periodic carriers who alternately gave negative and positive results on bacteriological examination of throat swabs for meningococci.

DR. MICHAEL FOSTER.

He proposed to give the Section a short sketch of the disease as it had been met with at the First Eastern General Hospital, Cambridge. Early in the history of the epidemic, the Commanding Officer set apart a ward for the treatment of the disease, and for it cases were collected from practically all parts of the Eastern Counties and South Midlands; at one time cases were drawn from eight counties. The first case came in the middle of January, an officer's servant, who had been at his work the night before, but at 2 a.m. became unconscious in bed. He was moved to the hospital, with a provisional diagnosis of uræmia. On arrival, he was profoundly unconscious, had tremors all over, he could not swallow, had nystagmus, and retention of urine. With a catheter only $1\frac{1}{2}$ oz. of urine could be obtained, and as it contained neither sugar nor albumin, lumbar puncture was performed, and 1 oz. of yellowish fluid containing pus was drawn off. It was submitted for diagnosis, and as the report had not been received next day, puncture was repeated, $1\frac{1}{2}$ oz. of fluid being withdrawn. On the third day the patient was definitely better. A third puncture was done, and after that he was able to swallow and had recovered consciousness. On the sixth day he was well, and was discharged, and on the twentieth day he returned to duty. Further cases came from the same regiment in a day or two; the first two of these were lumbar-punctured and both recovered, though their history was a somewhat tedious one. Both returned to duty. Then they began to employ serum, and they treated six cases of their own, and

two cases were sent to them from outside, after having already received an injection of serum. Of the first eight cases, four died. The conclusion formed was that they were not doing very well at that hospital with the serum, especially when the striking results which mere lumbar puncture had yielded were remembered. Succeeding cases were therefore dealt with in that simple fashion, the procedure being repeated as long as the symptoms persisted. To the end of March, twenty-eight cases of the disease had been treated there—i.e., twenty-six in hospital, two outside. Excluding the cases which had had serum, there were twenty cases treated with repeated lumbar puncture, with four deaths. But Nemesis was hard upon their heels, for five of the next seven cases died. That was during April, when there was a particularly virulent type of case. Towards the end of the epidemic, they had a series of nine cases, with one death, he believed. During the time he had charge of the ward—i.e., till the middle of June—there were forty-two cases of the disease, with fourteen deaths.

He wished to put before the meeting the question how far treatment by drainage and lumbar puncture, frequently repeated and persisted in during the continuance of the symptoms, was a satisfactory form of dealing with the cases, at least until something better in the way of a serum was obtainable than seemed to be the case now. The advantages of the treatment by repeated puncture were relief of the symptoms in a striking way, and particularly the lessening or stoppage of the severe headache. One case, which came in with the most severe headache of any they had seen, so that he rent the air with his cries, was submitted to puncture daily, and with very marked relief and subsequent cure. He had another case, treated with serum, which also recovered and enabled a comparison to be made. Another and most important point was the possible prevention of the occurrence of hydrocephalus in these patients. Of course, there was always the fulminating type of case which probably would not get better. Three of their cases came in unconscious, with inability to swallow, with retention of urine and rattling in the throat, and all three recovered in from five to eight days, after having had only daily lumbar puncture. Some cases went definitely downhill, despite the puncturing. He could not claim any extensive personal knowledge of the disease, though he had occasionally seen examples of it before this epidemic; yet after moving about among cases for five months one could claim a fair clinical acquaintance with it, and he was sure there was a type of

case which recovered to a certain point and then relapsed, and though the patients might linger, perhaps for weeks, having varying degrees of unconsciousness, yet death was the sequel, and then there was found to be extreme dilatation of the ventricles, and the cord was tightly bound across by thick lymph so as to prevent circulation. One such case which they treated with serum got worse, and died in a very short time. Another case came in and showed very much the same symptoms which others did, but subsequently developed hydrocephalus. Eventually, however, the patient got well and returned to duty. He had had very regular lumbar puncture. He exhibited that patient's chart. In several cases he thought a serious omission consisted in not having done lumbar puncture often enough and continued it over a sufficiently long time. Several patients suddenly lapsed into an adynamic state, with profuse sweating, later becoming unconscious, with a feeble running pulse, and ultimately dying of hydrocephalus. They were thought to be too weak to stand interference. Subsequent experience proved this ought to have been undertaken.

With regard to the serum treatment, in one of the later cases, which was running a very chronic course, as the patient did not seem to be getting better, 5 c.c. of his own serum were injected, after which he ceased to have headache, and his temperature fell to normal. He insisted that lumbar puncture was a very safe proceeding. He considered it very important to puncture at least the acute cases until acute symptoms subsided. This prevented the fluid becoming stagnant in the spinal cord, and the onset of a fatal result. In an acute case no one could foretell what would happen. Read in the light of subsequent experience lumbar puncture should have been persevered with. He agreed with the remarks of Dr. Robb as to the advisability of giving a general anæsthetic. It was difficult to do lumbar puncture and run off as much fluid as one desired without inducing general anæsthesia; moreover, without the tapping one was apt to break the needle on account of the violent movement of the patient. With regard to the quantity of fluid withdrawn, they had never witnessed any signs of collapse, such as changes in the pulse-rate, however much fluid came away, sometimes as much as 2 oz. 6 dr. A further advantage attaching to the anæsthesia consisted in the patient obtaining several hours' sleep: he generally had an interval of some twenty-four hours before the headache recommenced.

Captain J. F. GASKELL.

He merely wished to go into the cases at the First Eastern General Hospital with a little more pathological detail than Captain Foster had done. The cases which had died would first be dealt with (*see* Table I, p. 20).

Two cases, Bd.(1) and Hs.(1), were of the fulminating type, one lasting thirty-six hours, the other less than twenty-four hours. By the time the first of these patients was received he was moribund. He was given 20 c.c. of Mulford serum as only 20 c.c. of cerebrospinal fluid could be withdrawn. No effect was seen. The seven other cases in the group with no response to treatment lasted less than five days. In most of the cases it was fairly easy to ascertain the duration of the illness as the onset was sudden. In these irresponsive cases, the pus was not distributed mainly over the base of the brain, but there was a most intense vertical meningitis; the whole of the meninges were greatly engorged, and even in the early fulminating cases there was a large collection of pus all round the cerebral vessels; in most much change in the base of the brain was markedly absent. Pathologically, one might consider that this type of case was almost unsavable. The clinical effect of lumbar puncture in them was *nil*; in three serum was given, but with no effect. Every day lost from the commencement of the illness before lumbar puncture was done seemed to lessen the chance of recovery. Five of the cases were not punctured until the third day, and it was conceivable that if lumbar puncture had been done earlier, the cases would not have been so severe, and there might have been a chance of recovery.

A second group was represented by two cases, Ss. and An.(2). One case was punctured every day, and died on the nineteenth day of the illness. The amount of pus progressively increased through the whole series of punctures until about the ninth day; then it suddenly ceased, and there was nothing obtained but a thin serum. A large trocar was used, but even that was not of any use. The post-mortem findings afforded the explanation: the whole base of the brain and cord were matted with extremely thick pus, which could not be shifted with anything short of a scalpel. In this case serum was tried in order to thin the pus on the seventh day, but observations made did not show that it produced any effect.

A third group were cases which developed hydrocephalus—As., Dk., and Ye. The case Ye., he felt sure, was not punctured sufficiently

TABLE I.—DIED.

Name	Number of punctures	Days of illness on which lumbar punctured	Melford serum given	Pus	Number punctures showing growth	Days of illness on which they grew	Length of illness	Course of illness
Bd. (1)	1	2	—	Pus V.	1	2	36 hours	Fulminant
Hs. (1)	1	1	1 (20 c.c.)	" V.	1	1	36 "	"
An. (1)	1	3	—	" V.	1	3	4 days	Irresponsive
Ck. ...	2	3, 4	3, 4 (15 c.c.)	" V.	2	3, 4	4 "	"
Ja. ...	3	3, 4, 5	—	" V.	3	3, 4, 5	5 "	"
Dn. ...	3	2, 3, 4	—	" V.	3	2, 3, 4	5 "	"
Fx. ...	2	3, 4	—	" V.	2	3, 4	5 "	"
Ht. ...	2	4, 5	—	" V.	2	4, 5	5 "	"
My: ...	4	2, 3, 4, 5	3, 4, 5 (15 c.c.)	" B.	4	2, 3, 4, 5	5 "	"
Mt. ...	0	—	—	" V.	0	—	15 "	"
Ss. ...	9	4, 5, 6, 7, 8, 9, 11, 13, 14	7 (15 c.c.), 14 (6 c.c.)	Pus + + + +	9	4, 5, 6, 7, 8, 9, 11, 13, 14	19 "	Some response
An. (2)	15	3, 4, 5, 6, 7, 8, 9, 10, 11, 13, 14, 15, 16, 17, 18	—	Pus + +	15	3, 4, 5, 6, 7, 8, 9, 10, 11, 13, 14, 15, 16, 17, 18	23 "	Responded fairly
As. ...	14	4, 21, 22, 23, 24, 25, 26, 28, 30, 33, 35, 44, 46, 47	—	Pus + H	11	4, 21, 22, 23, 24, 25, 26, 28, 30, 33, 34	49 "	Responded well; relapse twentieth day
Dk. ...	30	4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 20, 23, 26, 28, 31, 32, 34, 35, 36, 37, 38, 39, 42, 44, 49	Subcut.: 14, 15, 18 (30 c.c.); 21, 22 (25 c.c.); (B. W. & Co.)	Pus - H	16	5, 6, 7, 8, 9, 10, 15, 16, 26, 28, 32, 34, 35, 36, 37, 39	51 "	Irregular; slight temporary response
Ye. ...	15	6, 9, 11, 13, 40, 43, 44, 46, 47, 48, 49, 50, 51, 54, 56	—	Pus - H	2	11, 13	63 "	Response very slight

H = hydrocephalus of third and fourth ventricles.

V = vertical; B = basal. Number of + signs = amount of pus. Pus - = no pus found post mortem.

TABLE II.—RECOVERED.

Name	Number of punctures	Days of illness on which lumbar punctured	Mulford serum given	Number punctures showing growth	Days of illness on which they grew	Length of illness	Course of illness
Tn. ...	2	1, 2	—	2	1, 2	11 days	Uninterrupted recovery
Rk. ...	3	1, 2, 3	—	3	1, 2, 3	11 "	"
Hs. (2)	5	4, 5, 6, 7, 8	—	1	4	15 "	"
Tr. ...	7	1, 2, 3, 4, 5, 6, 10	—	1	1	15 "	"
Wg. ...	1	5	—	1	5	21 "	"
Wd. ...	11	5, 7, 8, 9, 10, 11, 12, 13, 14, 16, 30	—	9	5, 7, 8, 9, 10, 11, 12, 13, 20	22 "	"
Yg. ...	10	5, 6, 7, 8, 9, 10, 11, 14, 17, 22	—	5	5, 6, 7, 8, 10	22 "	Practically uninterrupted recovery
Wr. ...	6	3, 10, 12, 15, 17, 19	3 (30 c.c.)	1	19	26 "	Fair recovery
Tr. ...	6	4, 5, 6, 7, 8, 9	—	6	4, 5, 6, 7, 8, 9	27 "	Uninterrupted recovery
Me. ...	1	3	3 (5 c.c.), 4 (15 c.c.), 5 (10 c.c.), 6 (15 c.c.), 11 (5 c.c.)	0	—	28 "	"
Bd. (2)	5	3, 4, 5, 6, 11	—	2	3, 4	30 "	Fair recovery
Gn. ...	—	—	—	—	—	32 "	Uninterrupted recovery
Sl. ...	8	4, 5, 6, 7, 8, 9, 10, 12	—	7	4, 5, 6, 7, 8, 9, 10	36 "	Fair recovery
Ns. ...	4	3, 4, 5, 9	—	3	3, 4, 9	38 "	"
Sm. ...	1	7	—	0	—	41 "	Moderate recovery (heart)
An. (3)	12	5, 6, 8, 9, 11, 15, 16, 18, 26, 35, 36, 38	16 (10 c.c.), 18 (15 c.c.), 26 (15 c.c.)	6	6, 8, 11, 15, 16, 18	48 "	Gradual recovery
Rs. ...	2	35, 49	—	1	35	56 "	"
Ja. ...	17	8, 9, 11, 12, 14, 16, 18, 21, 22, 27, 35, 39, 41, 42, 44, 48, 51	—	1	21	62 "	Intermittent recovery
Mw. ...	2	43, 60	—	0	—	64 "	"
Bn. ...	2	11, 12	—	1	12	72 "	Intermittent and prolonged recovery
Ty. ...	6	3, 9, 48, 67, 74, 77	77 (5 c.c.) of own serum	4	3, 9, 48, 67	86 "	Intermittent and prolonged recovery
Rn. ...	32	5, 6, 7, 9, 10, 11, 13, 14, 15, 16, 23, 25, 27, 29, 31, 32, 33, 34, 36, 37, 38, 40, 41, 42, 43, 46, 48, 50, 52, 56, 58, 60	—	22	5, 6, 7, 9, 10, 11, 13, 16, 27, 29, 31, 32, 33, 34, 36, 37, 38, 40, 41, 42, 43, 52	100 "	Intermittent recovery
Jn. ...	12	8, 9, 10, 11, 16, 18, 19, 22, 24, 31, 37, 43	—	6	9, 10, 11, 31, 37, 43	102 "	Intermittent and prolonged recovery

Black figures show punctures performed before admission to the first Eastern General Hospital.

often. The man was not brought to Cambridge until the sixth day of the illness, and he finally passed into a chronic hydrocephalic condition. The last puncture was on the fifty-sixth day, and then puncture was stopped. From the fortieth day he remained in an adynamic state, though his pulse was fairly good, and he was taking some food. The last seven days he had no puncture. Post mortem, a large accumulation of fluid was found in the third and fourth ventricles. This kind of case always died of respiratory failure. The patients could be kept alive for hours by artificial respiration, and so long as that was done the heart kept going. The interpretation of that was that there was pressure on the fourth ventricle, producing the respiratory failure.

At Cambridge there were twenty-three recoveries and fifteen deaths. Table II (*see* p. 21) gave the recoveries. The first two cases, Tn. and Rk., were punctured on the first day of the illness, and the duration of their illness (eleven days) was the shortest met with at that hospital. They responded very quickly, and a few punctures sufficed to start them on the way to recovery. In the chronic case of Ty., he was given 5 c.c. of his own serum, as this proved to be much more strongly agglutinative than the best of the other sera tried, including some obtained direct from Dr. Flexner at the Rockefeller Institute. The effect of this was to bring about convalescence. As another man's illness suddenly cleared up on the sixtieth day, too much stress could not be laid on this recovery—it might have been a coincidence. There was now in hospital at Cambridge a patient whose first day's illness was July 21; he was not admitted till six weeks later, because the condition had not been diagnosed. He was in an adynamic hydrocephalic condition, and it was proposed to try a similar treatment in his case.

The scanty particulars given tended to show that cases could be just as well saved by daily lumbar puncture and evacuation of fluid as by giving serum. At any rate their results, 39 per cent. mortality, were as favourable as those of others who had recently had cases of the disease. They had not given soamin or other substance; and it was their view that even when serum was given much of the benefit was attributable to the fact that cerebrospinal fluid was withdrawn in order to admit the serum.

Dr. CAIGER,

in response to Dr. Hale White's invitation, said that he proposed to mention a few impressions he had gathered as a result of the treatment of a number of cases of cerebrospinal fever which had been received into the South-Western Fever Hospital during the late winter and spring of the present year.

Though greatly interested in the disease, he feared he was unable to adduce anything very novel, either in principle or method of treatment. From the middle of February to the end of June, seventy-two cases had been sent up to the Hospital certified as suffering from cerebrospinal fever. Of these, twelve were obviously the subject of some other disease. In ten, all of whom recovered, the diagnosis was not confirmed by examination of the cerebrospinal fluid, and so the nature of the illness must be regarded as doubtful. In the remaining fifty, however, the true nature of the disease was confirmed by bacteriological, cytological and naked-eye appearance of the fluid drawn off by lumbar puncture. Of these fifty cases, sixteen died, a mortality of 32 per cent. All were treated with antimeningococcal serum. In the large majority of them Mulford's serum was used, but a few of the earlier cases were treated with a special serum, which Professor Martin had kindly placed at Dr. Caiger's disposal from the Lister Institute. As to the influence of the serum as a curative agent, he had no doubt whatever.

It was satisfactory to learn from Dr. Robb that he was in favour of giving a general anæsthetic for the purpose of lumbar puncture and the introduction of the serum, and that he had not encountered any ill-effects in consequence. Their own practice had been to administer chloroform in all cases except in adults with well-marked power of self-control, or when the patient was more or less comatose; and neither he nor his colleague, Dr. R. L. Wilcox, who was in immediate charge of the cases, had seen any reason to revise their views on the subject. Like Dr. Robb, he had ventured to ignore the necessity for simultaneous observation of the blood-pressure when performing the operation, as insisted on by Sophian, nor had they had any cause to regret it. That variations in the pulse tension and frequency occurred during both the withdrawal of the cerebrospinal fluid and the introduction of the serum was undoubted, but they were convinced that such variations occurred less frequently and were less pronounced under anæsthesia than without it. Having regard to the mental disturbance

and actual pain involved, and the impossibility of feeling certain that the patient might not suddenly become refractory, he regarded the administration of a general anæsthetic as desirable in most instances. The only occasion on which any serious development occurred amongst some 200 or more operations was in the case of a girl, aged 17, in whom the breathing suddenly failed, and artificial respiration had to be kept up for several hours in order to maintain life; but in this particular instance no anæsthetic had been given.

With regard to the dosage, the amount usually injected was 15 to 30 c.c., repeated after an interval of twenty-four or forty-eight hours, the number of subsequent doses being determined by the degree of amelioration of the symptoms and the condition of the cerebrospinal fluid. Care was always taken not to inject quite as much serum as fluid removed, and in a few cases in which the cerebrospinal fluid obtained was small in amount at a time, but little serum could be given on that occasion. In recent cases, with the object of obtaining a more powerful effect, they had allowed the injected serum, with the addition, no doubt, of a certain residuum of cerebrospinal fluid, to run out through the needle after the lapse of a few minutes, and had then injected a further quantity. The foot of the bed should be raised a foot or so off the floor during its introduction, and the head of the bed raised in turn while the fluid was escaping. This might be repeated a second time, the whole proceeding thus representing a sort of intraspinal "lavage." The results so far had been encouraging, and the method was worth a further trial.

Whether the serum were given by means of a syringe or by the gravity method appeared to him immaterial, provided care were taken not to inject too quickly. In the large majority of their cases a syringe was used for the purpose. He was convinced that the risk of injecting the serum too quickly, however, had been overstated. Provided that not more than 15 to 20 c.c. were introduced, five minutes would be sufficient time to allow for the injection.

The likelihood of meeting with a "dry tap," though sometimes, no doubt, dependent on the thickness of the fluid itself, was more often due to want of skill or experience on the part of the operator. In some cases, although the needle might have been introduced in the right direction, and to a sufficient depth, no fluid was obtained and the result proved disappointing. Just as the successful passage of a metal sound might call for the *tour de maitre* on the operator's part in the field of urinary surgery, so would a successful lumbar puncture sometimes

depend upon the possession of an educated touch by the hand that wielded the needle.

The want of success with the serum treatment in the various naval units, as evidenced by the results collated by Surgeon-General Rolleston, was distinctly disappointing. Assuming the dosage to have been adequate, one was inclined to regard a lack of consanguinity between the infecting strain and that from which the serum was prepared as the most likely explanation of failure, in view of the success which had attended the use of the serum in so many other quarters.

Of lumbar puncture alone, without injecting serum, he could not claim any experience. The results recorded in forty-two cases so treated by Captain Michael Foster were deserving of careful consideration. The reduction of intraspinal pressure in most cases of meningitis, meningococcal or otherwise, might be expected to relieve symptoms. It was possible that the best results might be attained by a combination of the serum treatment with a final abstraction of fluid—in other words, a daily intraspinal lavage, followed by partial, if not complete, draining off of intraspinal fluid before removing the needle. He would certainly give the method a trial when opportunity offered.

As to drugs supposed to exercise a curative effect, his knowledge was very limited. Soamin he had never tried, and urotropine appeared to be quite useless in their hands, even when given in full doses. He would like, however, to draw attention to the value of a mixture of chloral and bromide as a nerve sedative, when given in sufficient amount and kept up continuously. It was useful in dulling headache and reflex excitability, and inducing a condition of somnolence which was most helpful. The tolerance to these depressants exhibited in cerebrospinal fever was very striking. A dose of 20 gr. of each, repeated every four hours, might be kept up for a week or ten days, or more, with nothing but advantage. In three cases, it was true, a bromide rash appeared, but in practically all cases the mixture had been given as a routine treatment.

In conclusion, he would like to emphasize the paramount need for long-continued care in the after-treatment of a patient who might appear to have completely recovered from cerebrospinal fever. Medical officers responsible for military units should recognize the fact that, once a man had been the victim of a pronounced attack, that man was a damaged man, both physically and mentally, and that no matter how well he might appear to be, he was unfit, and for a long time afterwards would be liable to "crock up" if subjected to physical stress or undue exposure.

Were this fact recognized, as it ought to be, a good deal of hardship and disappointment would be avoided. No man should be certified for "active service" for at least a year after undergoing an attack of cerebrospinal fever, and in most cases it would be wiser permanently to invalid him out of the Service.

Dr. MACNAUGHTON-JONES.

This discussion brought to his mind an epidemic—occurring, he thought, in Cork about 1870—of what they then knew as "black spotted fever," or cerebrospinal *arachnitis*. Co-existent there was a severe epidemic of malignant scarlet fever. The cerebrospinal cases were mostly amongst children, and were almost invariably and rapidly fatal. The prodromal period, before the petechial spots appeared, was often only of some hours' duration. Very rapidly drowsiness, unconsciousness, and coma followed—rarely convulsions. Two cases occurred in the Poor Law Dispensary District, to which he was then Medical Officer. A child had died of malignant scarlet fever. He had the house disinfected as far as he could, and the rooms limewashed. He was called in to see another child who was ill of the same disease, and found two of the elder girls engaged in the limewashing. They were then apparently in perfect health. The next day he was summoned to the house, and found both in bed, evidently attacked with cerebrospinal fever. He had them at once removed to the fever hospital. Both were dead within twenty-four hours.

He determined to have a post-mortem on these cases as far as he could venture without the knowledge of the friends. Accordingly he went to the hospital the following day, and, with the assistance of the resident medical officer and a friend, he opened the spinal column in both cases to investigate the condition of the cord. In both they found dark, sanguineous fluid under the membrane in different parts of the cord. He closed the sections as carefully as he could, replacing the bodies in the very thin coffins which had been sent. Unfortunately some fluid oozed out and soaked through the joints of the coffins. The result was disastrous, as there was a great prejudice against any post-mortem examinations. When the friends came to remove the bodies, they suspected that they had been tampered with, and insisted upon seeing them. They had an infuriated audience outside the hospital gate that threatened dire vengeance on the institution, but though he had to try to clear his character to the committee, nothing came of it except a permanent impression made on his mind of his first and last experience of cerebrospinal meningitis.

Therapeutical and Pharmacological Section.

President—Lieut.-Col. W. HALE WHITE, M.D.

(January 18, 1916.)

DISCUSSION ON THE SOLDIER'S HEART.

Opened by Sir JAMES MACKENZIE, M.D., F.R.S.

WE all recognize that the trying life of soldiers, exposed to many and varied vicissitudes, puts a strain upon the heart. This strain inevitably finds out the impaired hearts, whether the impairment gives rise to physical signs of lesions or not. This is well understood and, in treatment, presents no feature which has not been recognized in civil life, and about this class of case I do not intend to say anything.

These cases, however, form but a small percentage of the cases that are actually invalided on account of their hearts. It has long been recognized, particularly since the instructive account published by physicians during and after the American Civil War, that there is a form of heart trouble to which soldiers are particularly liable, and which has gone under the name of the "soldier's heart," or the "irritable heart of soldiers."

Shortly after the outbreak of this War, soldiers invalided on account of heart trouble began to appear in my consulting room. These were few at first, but, as time went on, they came in increasing numbers, and I took great pains to find out the circumstances and signs connected with their complaints. These observations brought to light what seemed to me the main cause of the illness which affected the soldiers, so that when I was asked to take charge of the inquiry into disorders of the heart for the Medical History of the War, I had before me an object which would supply our researches with a very profitable aim.

In this work I have been associated with Dr. Wilson, and we have examined about 400 cases. In at least 90 per cent. of the cases that have been certified and treated as heart affections, we find that they are not primarily heart cases, and that the principles of treatment applicable to these cases are widely different from those which are applicable to heart failure.

SIGNS AND SYMPTOMS.

When we see the soldiers in this country invalided because of heart trouble we find a good deal of variation in their appearance and symptoms. The face is often lined and drawn; many are spare and thin with a great vasomotor instability, as shown by the manner in which the peripheral circulation varies—the hands and fingers at times going pale and cold; at other times the fingers are thick and red, and the nose likewise becomes red and even blue with slight exposure to cold. If they have been treated for some months by rest and feeding, some become pale, fat, and scant of breath.

The chief complaint is an absence of the feeling of being well—they often feel out of sorts; "rotten" is a term frequently employed. A sense of fatigue or exhaustion easily induced is common to all. Breathlessness on moderate exertion is frequent; pain over the region of the heart less frequent. The physical signs are variable. The heart's rate is often not increased, in some it is persistently increased, as frequent as 120 per minute. More frequently at rest the rate may be quite moderate, but exertion, sometimes slight, may produce an undue rapidity, and it is in consequence of this excitability that the term "irritable heart" has been used. Murmurs, systolic in time and heard in different regions, are frequent, while an increase in size, usually slight, is not uncommon. In a few cases there is a slight œdema of the legs. The mental condition is somewhat varied. Periods of depression are not infrequent, and the patients are often very irritable. They accept the view that they have something wrong with their hearts, and readily yield to all restrictions and are often content to lie in bed and brood over their woes.

These are the salient features in a great majority of cases, and you will recognize them as common in civil life. We find identical conditions, for instance, in people recovering from an exhausting illness, such as typhoid fever or influenza, or after a severe surgical operation. We see all these phenomena well marked in people who are suffering

from some infective organism, and we also find them in people who have suffered a long mental and physical strain, particularly with insufficient sleep, as in a daughter who has for long periods nursed an ailing mother.

The recognition of the character of these complaints is a guide to us in seeking for the cause of the exhaustion in these invalided soldiers, and in a great majority of cases we get a history pointing to an illness which preceded their breakdown.

ONSET OF SYMPTOMS.

The account given of the onset of symptoms is peculiarly instructive. They will say they were in the trenches and felt well and fit, until one day they felt seedy and ill, and this continued until they were compelled to seek medical advice, when they were found to have a raised temperature. A few days' relief was obtained, and they returned to their work still feeling far from well, and, after a few weeks of the strenuous life in the trenches, they collapsed, sometimes with loss of consciousness, breathlessness, or even pain.

The story of the onset varies: sometimes it is an attack of diarrhoea, which persists for a time; sometimes it is after a definite illness, such as measles; but most give a history which we can safely surmise as being due to an infection. In a few cases one cannot get such a definite account of the starting of the illness, but many do recognize the gradual onset of the trouble. That is the story of the majority of cases, but there are a number from whom we can get no suspicious history of infection, but where there is an account of a very strenuous life.

To grasp fully how this exhaustion is brought about, we must understand the life in the trenches. The story of some of these soldiers is illuminating. One in December, 1914, suffered from appendicitis, and was operated on. He returned to duty three months after, and in June went to the Front. Immediately on arrival he went into the trenches and was there a fortnight. Every night was spent in repairing the damaged parapets. They were constantly being shelled. He never slept at night, and occasionally got a few hours' sleep in the day—often being twenty-four hours without sleep. One day a shell exploded in the trench, knocking him over and rendering him partly unconscious. On regaining consciousness he stuck to his work for twenty-four hours, but had to give in, feeling weak and ill, with pain over the region of the heart.

EFFECTS OF STRAIN DURING INFECTION.

So great is the mental strain and bodily exertion, with sleepless days and nights, while the trenches are frequently bombarded, that one might be disposed to consider that these factors would be sufficient to account for all the symptoms. But we find identical symptoms present in many who have never been to the Front and who have had no excessive bodily or mental strain, but who have suffered from some febrile infection. The majority also, as I have said, give a history of some previous illness, and it is necessary that we should recognize the effects of strain or effort complicated by febrile illness. For that purpose I recite to you the following experience: A young officer was training for a foot-race, and every day spent some time running round a track. One day, while doing this, he collapsed, fell, and was picked up partly unconscious. A doctor was summoned, who said he had strained and dilated his heart, and that was the cause of his collapse. I was asked to see him and, after getting this account, I recognized that a man who had taken a considerable amount of exercise the day before could not possibly suffer from heart failure, so I asked him if he felt quite well before he began to run and he admitted that he felt rather seedy, but hoped to shake off the feeling with the exercise. The temperature was taken and found to be raised a few degrees, so I had no hesitation in stating that the cause of collapse was the toxic influence of some microbic invasion, and that, with the subsidence of the fever and a few days' rest, he would be all right. That is what happened, and he was able to resume his training and ultimately ran his race in perfect health.

The action of toxins produced in the body by bacterial invasions has not yet been clearly worked out, but so far as I have gone into the matter a clear and distinct group of illnesses can be attributed to the toxins even when the organisms themselves do little damage. The phenomena I have described as characteristic of the soldier's heart will give you an idea of the point I want to establish, that the condition is one of general exhaustion and the circulatory symptoms are but parts of a general manifestation. To appreciate this more clearly, so that we may separate them from those in which there is an actual lesion of the heart, I will call attention to some striking differences between the symptoms of the poisoned heart and the symptoms of the infected heart—i.e., where the organisms have invaded the heart—and of chronic disease of the heart.

(1) *From Infected Heart.*—In some cases there may be doubt as to whether the condition may not be due to an infection of the heart itself. It may not be possible to settle the question while there is a rise of temperature, so judgment will have to be deferred until the fever subsides. If the temperature remains raised for a period of weeks, and no source of infection can be found, then the suspicion that there is an affection of the heart becomes strengthened. With the permanent fall of temperature, the heart can usually be assumed to have escaped infection and the abnormal manifestations (increased rate, systolic murmur, and increase in size) may be taken as the reaction of the heart to a toxin, or to the factors that produce a general exhaustion.

(2) *From Chronic Heart Disease.*—The first sign of a failing heart affected by some old-standing lesion is invariably a subjective one, due to a distressful sensation arising when some effort is made which the individual used to perform with no distress. By far the most common sensation occasioned in this way is breathlessness, the next most frequent, pain. Both these may be associated with palpitation. These are sensations which everyone recognizes as indications of inefficiency of the heart. In the poisoned heart with general prostration these are usually not the first signs, though they also appear, the first sign being the sense of exhaustion.

THE SENSE OF EXHAUSTION.

This sensation is often taken as a sign of cardiac inefficiency, but it owes its causation to a different mechanism. When people suffering from weakness consequent upon an infection, or after an exhausting illness, attempt to walk on the level until they are conscious of their weakness, the sensation by which they perceive their weakness is one of exhaustion or sometimes of giddiness. If they stop, sit, or lie down the sensation speedily disappears. If they attempt a more violent form of effort, such as walking quickly, or up a hill, or running upstairs, they may be pulled up by breathlessness or palpitation—sensations provoked by heart weakness. It is to the sense of exhaustion and its allied sensations, giddiness and faintness, that I wish to call attention, because the mechanism of production is different from that producing the sensations in patients with primary heart failure.

In people suffering from some toxic influence, the central nervous system is always affected. This is shown by the sense of feeling ill, by depression and irritability of temper. There is also evidence of vasomotor

disturbances, as shown by susceptibility of the peripheral circulation—the hands and feet persistently cold, or made so by excitement. There is a persistent over-action of vasomotor influences, like a sense of chilliness that persists after a cold bath; at other times flushes of heat pass over the body, and warmth may tend to overfilling of the peripheral vessels. This is seen in people who faint when standing in a warm room, or who speedily become exhausted or even faint when exertion is made. In these the blood tends to accumulate in the peripheral veins of the limbs and in the large abdominal veins, with consequent anæmia of the brain. It is because of this anæmia of the brain that the sense of exhaustion and syncope are provoked.

I do not enter here into all the facts which support this explanation of the sense of exhaustion, as many observers besides myself have fully dealt with the subject. My purpose here in drawing attention to it is to emphasize the mechanism of its production and to show that it is produced in a different manner from the signs of cardiac exhaustion, breathlessness, palpitation, and pain.

THE CHARACTER AND MEANING OF CARDIAC PAIN.

Another distinctive feature which is occasionally present is a sense of discomfort, amounting sometimes to pain, even of a severe kind, felt over the region of the heart. The pain may be occasionally provoked by exertion, but more frequently it comes on when the individual is at rest. Its occurrence, not in response to effort, is characteristic of pain due to a poisoned heart, for angina pectoris due to disease in the young occurs when effort is made. While not absolutely diagnostic, the occurrence of pain when this type of patient is at rest is strongly suggestive of poisoning.

HEART ABNORMALITIES ARE BUT A FEATURE IN A GENERAL CONDITION.

Taking into consideration all the facts, it will be found that the condition from which certain of these soldiers suffer, who are usually understood to have acquired a heart affection, is not, properly speaking, cardiac in origin but is the outcome of an injury to other systems as well as the heart, such as the central nervous system. Even when we find such marked abnormalities as increased rate, systolic murmur, and an increase in the size of the heart, the cause of these signs ought not

to be looked upon as disease, but merely as part of the manifestation of general illness. The importance of this point of view will be realized when we consider the treatment.

THE CAUSE OF ILLNESS.

While the reason for attributing the majority of these cases to bacterial and toxic influences is so far purely clinical, good grounds can also be found from bacteriological evidence. As, however, bacteriology is outside my province, I will not touch on that evidence further than to point out that I have frequently met with these phenomena in civil life in people in whom the causative organism has been detected. Here, however, I should like to point out that while the active agent producing these phenomena may be an organism or its toxin, or the products of physical exhaustion, some changes are produced in the economy—in the blood, central nervous system, or heart muscle—which impair the functional activity of these structures. I do not pretend to be able to decide which of these conditions is present, and in any case they do not materially affect the principles of treatment which have been found of value. It is possible, however, that where the illness is due to a microbe, vaccine therapy may be of value; but I am not qualified to speak of this.

NOMENCLATURE.

It is difficult to get a term which will convey clearly what the nature of the trouble is, so I use here the simple one of general exhaustion. It is a great misfortune that a custom has arisen that each sick soldier has to be certified as suffering from a definite complaint. Naturally, in the hurry and bustle of the earliest examination, the examining doctor has not the time to make a careful study of a case, as on their immediate breakdown the patients often present features of exhaustion in which the cardiac phenomena stand out most prominently, and the doctor who sees them naturally labels them by these phenomena. It is for this reason, no doubt, that so many of them are invalided on account of "valvular disease of the heart," "disordered action of the heart," or "dilatation."

My description, like my nomenclature, is very general, and as we inquire deeper into the subject we may be able to distinguish separate groups, each with a definite cause.

TREATMENT.

I have dealt at some length with the diagnosis of these cases, because, if we recognize the nature of the illness, then we can readily see on what principles treatment should be based. The lines are those we employ in civil practice. If the patient be the host of some microbe, the treatment should be devoted to increasing his power of resistance. If he be poisoned, treatment should be devoted to the elimination of the poison. Should certain tissues be injured by the invading microbe, or by the toxin or by physical strain, then treatment should be devoted to their healthy renewal. These indications are practically those which guide us in treating individuals after an exhausting illness. We have, however, in addition, to consider the mental condition of the soldier whose experiences have depressed his mental faculties and who has been told he has some heart trouble. This knowledge always tends to depress and to make them extremely amenable to that form of treatment that is the worst for them—rest in bed or the avoidance of exertion. If we recognize that such cardiac phenomena as murmurs, increase in size, and variability of rate, are but the evidences of an irritated heart, we shall see that too much rest is not beneficial. Every organ benefits by the judicious exercise of its functions, and the heart can also be beneficially stimulated.

The principles of treatment, then, should be devoted to increasing the health of the body as a whole in such a way as to increase the natural resistance to infection, to eliminate toxic influences, and brace up the whole man bodily and mentally. The best way to achieve this is by fresh air and judicious exercise in the fresh air. I need not labour the fresh air idea as it is evident to all, but I want to emphasize the question of exercise. There is a notion, deeply rooted, that if there is anything wrong with the heart the patient must be restricted in the amount of effort, and medical men who are themselves afflicted are quite content to lie in bed or move gingerly about.

The line I have adopted with those soldiers who have consulted me is that which I have adopted in civil life. All those I have seen have already been told that their hearts were affected, and this knowledge tended to depress. They often feel miserable, so that there is a mental side to the case, which is aggravated by the supposition that there is something amiss with the heart. This aspect of the case has to be met, and, consequently, in our treatment this view has to be kept in mind. When all fever has subsided, or when there is but an

occasional rise of temperature, I encourage the patient to get up and go about. I find out the form of outdoor exercise which gives him most pleasure. In my private practice my patients have been officers, so I recommend them to start as soon as possible any form of sport or game which they can get—fishing, riding, shooting, golf, &c. In recommending such exercise, I have two objects in view. The first is the mental condition of the patient, and I need not insist upon the beneficial effects of occupation. The more interesting that is, the more likely is the patient to be taken out of himself. I have referred to the fact that the sense of exhaustion is the predominant symptom in these cases, and that it is probably vasomotor in origin, due to the irritation of the central nervous system. We know that the mental state may play a part in producing exhaustion, for we find that a boring form of exercise will readily produce it, while a form of exercise that is full of interest can be borne for long periods without any exhaustion. Therefore, this out-of-door occupation should be in an attractive form.

In recommending effort, I know it will be said that there is danger in this just as there was when the soldier was employed in his military duties, particularly when such physical signs as murmurs and dilatation are present. If the true nature of these signs be recognized, that they are but the expression of a temporary enfeeblement of the heart muscle, and that the same measure for restoring a flabby leg muscle applies to a merely flabby heart muscle, we shall see the importance of exercise judiciously employed. No doubt injudicious exercise may do harm, but there is one precaution of a very simple kind which suffices to prevent any danger, and that is to instruct the patient to indulge in his exercise so long as it gives him pleasure and causes no distress or discomfort, but to stop or slow down as soon as he experiences the sense of exhaustion, breathlessness, or pain. This simple and easily comprehended dictum contains in it the whole philosophy of the therapeutics of exercise as applied to the heart, and may be used with safety in all obscure and doubtful conditions of heart weakness.

It will, however, be said that it is all well and good for officers who can afford such means of pleasurable occupation, but what about the private soldier cooped up in the wards? It is for him I wish to prescribe. I used the officer to illustrate the principle so that I may strongly urge that the same principles should be applied to the treatment of the private. If the officer is depressed and makes but slow progress in his comfortable quarters, what of the private soldier in the gloomy wards, meditating on his troubles, or "grousing" with his fellows,

with an occasional joy-ride in a motor-car? I want this argument for congenial exercise to be fully considered, not only for the pleasure of the sick man but so that the soldier may speedily regain his health and return the sooner to his duties. I know that already in convalescent camps the aim of interesting occupation with fresh air is being carried out, and I recommend the same principle to be applied in the so-called heart cases. But in these exhaustion may arise too soon for the participation in mechanical occupation, and I would recommend the same kind of mental stimulus which has been found beneficial in the officer should be employed—that is to say, games. Games absorb the attention and infuse a different spirit from any other kind of employment, and a selection of some games, as bowls or quoits or skittles, would add materially to our therapeutic resources.

As a practical outcome of these suggestions, I would recommend that the Government should provide a large hospital for the reception of soldiers who are supposed to be suffering from a heart affection: that these principles of treatment I have detailed should be employed for the recognition of the nature of the complaint, and the principles of treatment here recommended should be carried out. As soon as signs of improvement are shown, the soldier should begin drilling, at first for a short time each day, and, as improvement goes on, the time increased until he is able to undertake a normal amount. In this way his preparation for fitness would begin early, and he would be rendered fit much sooner than under the present method of treatment. I would also suggest that this view of the nature of the soldier's heart should be communicated to the medical officers in order that the diagnosis should be in conformity with the actual condition, and in this way prevent the grievous injury that is done to the sick soldier by implanting in his mind the depressing idea that he suffers from an affection of the heart.

Dr. R. M. WILSON.

Sir James Mackenzie has just afforded you a picture of the condition known as "the irritable heart of soldiers," as he has seen that condition in the cases examined by him. It is not my purpose to add anything to what he has said, because a careful study of upwards of 200 cases of the condition has brought me to the same conclusions as those enunciated by him. Like him, I have met with what I can only regard as a definite clinical picture, sharply defined and differentiated. The cardinal symptoms which he has indicated—a sense of exhaustion, breathlessness on slight exertion, a rapid pulse which becomes much more rapid upon the slightest attempt at action, pain over the præcordial region or along the left costal margin, and a vasomotor condition of greater or less stability—have been present in all my series. I have observed, also, the tendency to attacks of giddiness to which he drew your attention, the nervous symptoms, the generally high blood-pressure, and the tendency to persistence of the condition so long as the soldier is kept confined to bed. I propose, therefore, to confine myself to a consideration of the history of the condition, which I have worked out in some detail, and I shall include under this heading the various theories regarding causation that have been advanced at one time or another.

You may, perhaps, know that in 1864 the British Government appointed a Committee to inquire into the heart conditions prevailing in the Army. The Committee was convened by Earl de Grey, and consisted of three generals and two doctors. It sat in the years 1864, 1865, 1866, 1867, and 1868, and issued four reports. In the last report the question of infantry accoutrements was considered, and a recommendation was made that the principle of the brace should be adopted. The idea was that the old form of accoutrement restricted the heart's action. The Committee described the condition of "irritable heart," which they seem to have recognized clearly, as "an extreme excitability of the heart, combined with some, but not great, enlargement. During rest a heart of this kind beats easily, but on the least exertion its action becomes irregular, and the man becomes breathless." The recommendation of the Committee regarding the change of accoutrements was later adopted.

The American Civil War, meanwhile, was furnishing a large number of soldiers' hearts, and in 1864 Henry Harthorne [3] published a paper in the *American Journal of the Medical Sciences*, which, I venture to say, furnishes an exact picture of the condition as Sir James Mackenzie has

described it to you to-day. Dr. Harthorne laid stress on the rapidity of the pulse, on the acceleration of the heart's movements on the slightest exertion, on the shortness of breath, and on the cardiac muscular weakness. He suggested that in the case of the soldier the heart was injuriously affected by long-continued over-exertion, with deficiency of rest and often of nourishment; and he pointed out that several months of rest and treatment in hospital failed to do more than improve, without really curing, a large proportion of well-marked cases.

Two causes had thus been suggested so far back as 1864—tight accoutrements and over-exertion. The tight accoutrements were done away with, but the "irritable heart" remained. I pass over here the contribution to the subject made by Dr. Arthur Myers [4], of the Coldstream Guards, in 1870, because his description is similar to that already given, and because he also regarded accoutrements as the exciting cause of the trouble. But I must call your attention specially to the paper by Da Costa [1], published in 1871 in the *American Journal of the Medical Sciences*. This paper is the best-known contribution to the subject. Da Costa saw upwards of 300 cases in a hospital in Philadelphia, and he supplies the following striking picture:—

"A man, three months or so on active service, was seized with diarrhœa, annoying yet not severe. Soon rejoined, and then noticed that he could not bear exertion as well as formerly. Out of breath and unable to keep up with his comrades; dizziness and palpitation. Accoutrements oppressed him, yet otherwise he seemed well and healthy. Sought advice; was sent to hospital, where his persistent quick pulse confirmed his story. Digestive disturbances, if present, passed away, but the irritable condition of the heart remained, and only very slowly did this get normal, or it failed to do so."

Da Costa then proceeded to paint the same picture as that which has been presented to you to-day by Sir James Mackenzie. He mentioned, however, that he had noted that intercurrent attacks of fever caused the pulse to become less and not more irritable, and to slow down while the attacks lasted.

In 1876 a new phase of the subject was presented when, the change of accoutrement having failed signally to effect any improvement, Surgeon Arthur Davy [2] suggested that what he called "setting-up drill" was the cause. His theory was an ingenious one. "Setting-up drill," by over-expanding the chest, caused dilatation of the heart, and so induced irritability. This view found support in 1880 from Brigade-Surgeon Veale [5], and in 1896 from Surgeon-Captain J. B. Wilson [6],

but the discontinuance of "setting-up drill" does not seem to have put an end to the condition. On the contrary, I have myself met it in cases where this cause could be absolutely excluded, the patients having undergone little or no drill.

The theorists, therefore, are bankrupt; the disease remains. More recently we have been asked to view the disease from the bacteriological standpoint. And more recently still, to view it as a condition depending upon faulty action or excessive action of the thyroid gland. I am not in a position to discuss these views, but it is, I think, interesting to note in this latter connexion that some eighteen of my cases, or about 1 in 9, have definitely enlarged thyroids, and that thyroid administration in those cases in which it was tried seemed to exacerbate the symptoms. The administration of X-rays to the thyroid gland is said to have effected improvement in some cases. Let me say, however, that I have seen cases presenting very much the same clinical picture in which the X-rays did not help matters at all.

In view of the dangers which seem to have attended the making of theories to explain this disease in the past, I am of opinion that we must preserve an open mind. There would appear to be but one sound method in regard to the condition—the method of prolonged observation and continuous study. Many tempting views suggest themselves, but few stand the test of further investigation. On the other hand, a few facts seem to have emerged. One of these is that a cheerful atmosphere does confer benefit and a depressing atmosphere does exaggerate the condition. The patients are always better when their minds are enlisted in some more or less congenial occupation.

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Dr. F. J. POYNTON.

The difficulty of the subject must make me, I fear, somewhat vague and discursive, but I have endeavoured to collect for this discussion my independent thoughts on the question. Our views on treatment must be influenced by those that we take as to causation and also by the particular mental picture we have formed of the clinical entity. My inclination at the present time is to look at "soldier's heart" from a wide standpoint, feeling that we have before us a collection of symptoms predominantly cardiac, which are prone to arise in presumably healthy men at a vigorous time of life as a result of the exigencies of military service. The first point that attracts me is that of latent infection—latent in the sense that the acute process is either spent or was never sufficiently obvious to attract ordinary attention. This view has been brought again into prominence by a recent memorandum of Dr. Cotton, Dr. Lewis and Dr. Tele. These writers directed attention to the presence of streptococci and staphylococci in the urine of such cases. They indicate their possible causal significance. As indirect evidence, I can state with confidence that the streptococcus isolated from acute rheumatism can produce, irrespective of endocarditis and pericarditis, cardiac failure in animals, with or without notable dilatation. From their urine the streptococcus can again be isolated and reproduce the symptoms. Two obvious difficulties must face these observers: (1) the proof that the micrococci found in the urines are also a cause of the cardiac weakness, and (2) an expression of my own experience—the fact that scanty cultures from urine are apt to be non-virulent, and thus to obstruct experimental proof. It is, however, clear that if the future establishes the reality of their kind, we must add to such well-known infections as the rheumatic, the influenzal, enteric, dysenteric, and others, a group which, for the moment, I will call "septic." Again as indirect evidence, I would emphasize a group of cases in the rheumatic—both child and adult—in which the heart is weakened without either endocarditis or pericarditis. A study of these in childhood has led me to see many symptoms similar to those occurring in "soldier's heart"—e.g., absence of organic murmurs, liability to failure on physical strain, vasomotor disturbances, dyspepsia, nervousness, and irritability of action.

If latent infection is the predominant factor, then the line of treatment is clear and is largely preventive—viz., it will be based on accurate histories, on the removal of obvious foci of infection in tonsils or teeth or elsewhere, and on patience with convalescence. If the

condition is established, rest and cautious training of the heart to increasing exertion. My opinion is that, in severe cases, we shall get forward to a certain point, but that this will be short of the requirements of active service.

Although, personally, I do not look upon these as the classical cases, I realize that, at the present time, the factor of infection is of the greatest importance. We have numerous soldiers in England recovering from enteric fever, dysentery, and gastro-intestinal disorders, and, theoretically, a potential army of "soldier's heart." Curiously enough, a remedy, quite unforeseen, has arisen in the elaborate tests required by the War Office to guard against the spread of infection. Three or more such tests on a large scale take time; and thus we are able, under military discipline, to guard these most rash of convalescents against their own destruction, and brace up their weakened hearts by prolonged convalescence.

What I call classical cases I have seen in the ten years preceding the War among the privates in our Guards regiments. They have seemed to me all of the same type. Adolescents of great stature and with big frames, often decidedly anæmic, they are examples of that condition which is called in domestic medicine, "outgrowing your strength." I need not give individual histories in detail. The symptoms commenced early in their training with great fatigue at the end of the day, then thumping of the heart at night, then dyspnoea and palpitation on exertion, and finally a fall-out dead beat from the ranks, or a sudden syncope, with or without some convulsive movements. All were nervous, the majority dyspeptic; some had pain. Most of them smoked cigarettes, possibly some were addicted to beer, but of this I was not convinced. In these cases the well-known forcible, ineffectual, ventricular beat was obvious. The pulse was small in proportion to the man and size, and very variable in rate, sometimes irregular. I do not think infection was a factor in these cases, though it is impossible to dogmatize upon this point. I regard the weakness of the heart either as congenital, or more probably, and this is more important, as a transitory condition due to the large, ill-consolidated frame having outstripped the cardiac development.

The elements conducing to strain are not far to seek. We all know their projecting chests—which closely resemble the breast of a thrush standing in the snow—with the consequent thoracic breathing and limited expansion of the diaphragm. The physical exertion under training is undoubtedly heavy, and they cannot take a day off when

they do not feel quite up to the mark. Dyspepsia increases their difficulties, and lastly, but not least, when the symptoms commence they often lose nerve, for such subjects are but ill-suited to suffer visceral discomforts.

I believe those cases which have appealed to me as classical are due to the premature over-exertion of frames imperfectly consolidated, and of hearts not proportionately powerful. Taking this view of the causation, I naturally consider that the great step in treatment is prevention, for when once the condition is established I am doubtful whether any treatment will bring them up to the required standard of active military service, though undoubtedly, in course of time, they recover sufficiently for a less exacting occupation. In a few years these young fellows would be fine men, in my opinion, and though I do not suppose barrack life can be conducted on the lines of an expensive preparatory school, I venture to think that perhaps we are a little too English in our methods. Trusting overmuch to the many good things Providence has given this nation, we possibly underrate our responsibilities, and are too satisfied with the attitude that "an unsound man has no use for us." Whereas the realization of the great value of such lives might lead to a more careful supervision of these young giants during their early training and guide them over a period of life when, though physically remarkable, they are still in reality delicate, because their development is not yet completed. I have had no military training in the regulation sense, but my experience of these cases leads me to think that more might be done to obviate these catastrophes by prevention.

The next aspect of the subject, upon which I would venture a few remarks, is that of nerve shock and strain. We all, no doubt, have private views upon the nervous system and the heart. To me this vital organ has always seemed almost a part of the nervous system itself. And no consideration of heart-strain bereft of the nervous aspect seems possible. The muscle may be there to contract, but if the nervous mechanism is disturbed, the results will be almost as unfortunate as if the muscle were damaged. In cases of "soldier's heart" both nerve and muscle are, I imagine, often at fault, and sometimes the muscle is the first sufferer, but in cases of nerve shock I think it is the nervous element that predominates.

Striking instances of the result of high explosives have come under my care. In such cases there had been no previous complaint of cardiac trouble; yet afterwards for months there had been palpitation, dyspnoea, rapid, variable pulse, vasomotor changes, and præcordial sensations; and

in disposition, loss of decision of purpose. The cardiac distress in some cases completely unnerved these patients. Preventive treatment is impossible here clearly, and the interesting question arises—upon which I hope to hear the opinion of this Section—as to whether any treatment will render such patients fit for active service in the near future.

One case bearing upon this point I should like to record briefly: A young officer, a hero of the great Ypres fight, powerful and fearless, was buried by a shell and invalided for nervous shock, in which cardiac symptoms, such as palpitation on exertion, tenderness over the apex, syncopal attacks, irregularity of action, vasomotor crises, and throbbing were prominent. There was no apparent valvular lesion, and there had been no obvious injury. He was sent to me by a doctor who inclined to the view that it was all purely functional, and that he had had enough of the War. I kept him quiet and obtained him prolonged leave, but he returned to the Front without my knowledge, in the middle of 1915, about ten months after his first collapse. He was in the fierce fighting close to Lille, and broke down at once with the same symptoms, predominantly cardiac, which reached such a pitch of acuteness that he practically lost control of himself in a furious outbreak of palpitation. When I saw him after this his cardiac symptoms resembled those observed on the first occasion.

The possibility of hyperthyroidism as a factor is very interesting, for it introduces the possible value of X-ray treatment. My own experience is perhaps exceptional, and I am very anxious to get the right perspective. In only one case have I noticed a large thyroid with fine tremor, but I at once admit that hyperthyroidism may occur with very little positive indication in the thyroid. Heat-stroke is yet another factor in some cases, and is exceedingly difficult to combat, as anyone who has suffered from cardiac symptoms from a slight touch of the sun will realize. When the symptoms are severe I am very doubtful of a sufficient recovery in a reasonable time.

In the treatment of these cases due to nerve shock there is no room for prevention, and I am in agreement with the general principles laid down, except that, unless for the purposes of research, I am averse to collecting cases of the same character either into special wards or special convalescent homes. A cheery doctor, a minimum of complete rest, bright companionship, pleasing exercise short of fatigue, and time are my mainstays. Care of the digestion and soothing drugs are of much value. Of special drugs I have tried tincture of *cactus grandiflora* in many cases without any untoward result.

Captain (Temporary) R. W. MICHELL, R.A.M.C.

Of late I have had good opportunity of observing soldiers in the early stages of inefficiency of the heart. The knowledge with which I approached the subject had been gained over a period of many years from the constant examinations of the young athletes in residence at Cambridge. Such as it is, I give you my experience. The soldiers were mostly young men, enlisted recently, who were carrying pack, rifle, and odds and ends intended to lessen the rigorous simplicity of trench life. Their inefficiency began as weariness, with shortness of breath, and was followed by greatly increased sensitiveness to the beating of the heart. There was no pain nor tenderness, and palpation, percussion and auscultation showed little, if any, enlargement beyond what one expects to see in any young athlete in full exercise. This state passed to one in which the veins of the neck were filled and continually and rapidly varying in distinctness of outline. Irregularly occurring murmurs could be heard over the "apex." The picture would have served for one of sudden partial exhaustion of the right side of the heart but for the absence in some of both pain and tenderness, especially tenderness. And, indeed, I have often had doubts, which have grown with the realization that both conditions can be present together. At present I know I cannot distinguish for certain between them in all cases unless I am able to watch the patient. A right ventricle ceases to be even an annoyance in, say, forty-eight hours; an irritable heart does not, but on the contrary becomes more insistent on the attention of the patient as its other symptoms and signs develop. Of these the most frequent and noticeable are dilatation of the pupils, a very marked sensation of sinking in the epigastrium, cold skin, and the passage of large quantities of very light-coloured water.

I have omitted to mention change of position of the apex-beat as a help in diagnosis, because it is unreliable, inasmuch as the heart-beat of a young man taking hard physical exercise may be seen to beat an inch or more further out after exercise without being in any way affected. In older soldiers the hard work also produces partial failure of the heart, but it seems to take another form, that of weakness, or weakness combined with dilatation. Sudden partial failure of the man is not infrequently due to sudden partial failure of the right auricle. These two classes, the young and the old, are not sharply defined, and there arises the question as to what it is which decides whether a man is to suffer from irritability or partial failure of the muscle. There is a

large number of adolescents "irritables," who began to be so in boyhood, who have had "attacks" on and off ever since, just as there have been boys with weak heart muscle who are now adolescents with weak heart muscle. There remain those who fall into neither of these classes, and who seem to acquire irritability. These are the cases which call for explanation. During our training in physiology we have heard that irritability of tissue and youthfulness go together. It is true, but it is not an explanation, and it does not at present appear to be the whole truth.

A common though not invariable accompaniment of a temporary partial failure of the cardiac muscle, with features of the irritable heart, is the partial failure of the chest wall to move to the usual amount and in the usual way. The healthy chest shows a constant relationship between its measurements at, for example, the level of the third space and the level of the xiphisternal joint. The measurement taken at the lower level during full inspiration is greater than that taken at the upper level in full expiration. When this relationship is upset, a history of some severe disease of the chest can be obtained, as a rule, from the patient, and if not from him, from the chest itself. The effect of hard exercise on the chest wall is to lessen the range of movement. This change takes the form of a gradual approach to the position of the chest in full inspiration. The measurement of the chest in expiration lengthens, while the measurement in inspiration falls—the first-mentioned alteration is the greater. This may go on until the chest is fixed in a position which is but a little short of the full inspiratory phase of its movement. In this case the heart may be for a time intensely irritable and somewhat inefficient. Recovery is rapid and complete when rest from hard exercise is ordered and the expiratory muscles are helped to empty the chest by external pressure. A patient of mine won a race at Henley Regatta just under six weeks from the day he greeted me with: "I've cracked—my heart's gone; I feel every single beat, and can't breathe better than this." Another played polo for the University of Cambridge five weeks after he had one day got off his horse because he felt as if he were about to be suffocated. He, too, felt every beat. It is at least worthy of mention that the true irritable heart of the soldier does not only occur in men afflicted as were these two I have mentioned, while the frequent painful inefficient heart does.

May I say in conclusion that I cannot believe fright has much to do with irritable hearts? If it had, the number of these would be much greater than it is. Doubtless the soldier's task of keeping himself in

hand and up to his work when in imminent danger is very exhausting, but I would point out that this exhaustion, which compels men at the Front to give in eventually, does not appear to be the source of supply of irritable hearts.

DR. PHILIP HAMILL.

I must say that certainly the experience of everyone who investigates these cases must closely agree with that of Sir James Mackenzie. Sir James limited himself to a very specific class, and I think that the more one sees these patients, the more one realizes that the true irritable heart of the soldier is a very definite condition. I have had cases returned as soldier's heart which were cases of congenital disease; there have been one or two cases of sub-aortic stenosis. I have had two cases of exophthalmic goitre, one only a mild case, with slight enlargement of thyroid. But we can put these aside, because they are not in any way different from the cases of heart trouble met with in civil practice. In most cases irritable heart can be traced back to some undue exertion during some febrile attack—it may be called a cold or influenza. Many own to some diarrhoea, but the issue is complicated by the fact that so many men have diarrhoea and not heart trouble. It is surprising, however, how high a proportion of soldiers with irritable hearts give a history of diarrhoea. I do not include in my class cases which have had a large number of true premature beats. I have taken pulse-tracings and electro-cardiograms of cases, and have been somewhat surprised to find that the latter are almost invariably normal; but in most cases in which I have taken the cardiograms, the patient has been a little excited, or has had to climb a flight of stairs before reaching the room, and then the pulse has been, and has long remained, too frequent for premature beats to be manifest. A common rate is 100; I have not obtained a record of a heart beating at 60 or 70. Generally the mere suggestion of the electro-cardiograph and the putting of the hands and feet into the electrode jar are sufficient to hasten the pulse, and it confirms the importance of the psychical factor in these cases; these people are readily upset by any slight excitement.

There is one man whom I have been able to study more closely than other patients. Some years ago he was a Territorial officer, and then joined the Regular Army, in a cavalry regiment; he was also a boxer. He had been a little unwell one day, when doing a long march with his company, and he fainted by the wayside, and had to lie

down. He recovered after a while. The same thing happened on another march. He was sent to hospital, and appeared before a Medical Board, but they could find nothing except some dilatation. The pulse-rate was not unduly high, and the Board passed him as fit for duty. But one of the senior medical officers told him it was unwise for him to continue in the Army. He is now a medical student, and I have had the opportunity of examining and observing him closely. He is by nature restless, and does not concentrate his mind on one thing for long at a time. He constantly thinks of his discomforts and disability. He has caused me some anxiety because he is anxious to rejoin. He went to attest, but was promptly rejected by the examining doctor because he had a slightly increased area of cardiac dullness, an increased pulse-rate, which reached 120, and gave a history of having had to leave the Army. He had at the time some œdema of the ankles, but has not had albuminuria. This student's symptoms were greatly accentuated after a bomb from an aircraft fell near his room. His pulse-rate readily rose to 140 per minute after moderate exertion, and remained high for a long time. He has lately been working hard for an examination, and his symptoms—breathlessness, præcordial pain, giddiness and faintness—have greatly increased. Hence he has recently become more irritable. That raises the question whether a man badly broken down on the march is ever likely to be fit again for heavy duty. My experience has been limited, and the opportunity I have had of studying cases for long periods has not been great; but my impression is that, however much these men may improve, they are always ready to relapse when the favouring circumstances recur.

With regard to the effect of fresh air and moderate exercise, my experience bears out that of Sir James Mackenzie. Probably the heart muscle has been strained in some way, perhaps over-distended, but nevertheless there is a considerable psychical factor to be treated before recovery can be expected. Games in the open air and pleasant forms of exercise are of great value, and considerable exertion can be undertaken if the mind is suitably occupied. Hence it is important that these men should be treated in a hospital with pleasant surroundings, open air and green fields, with ample opportunity and encouragement to take exercise. The same methods I have found of value in treatment of cases of shell shock. Quite apart from the psychical factor, I think that in a large proportion of these cases the heart is permanently damaged, and there is little likelihood of their ever being fit for the arduous duties of active service. It is for us to keep the names and

particulars of these men, and to follow them up. It will probably be for the next generation to draw conclusions as to the precise nature of the disorder.

Dr. ALEXANDER MORISON.

When I came here I was under the impression that one would hear more about the soldier's heart in a wider sense than we have done. We may conclude, from most of the remarks, that the one indubitable fact in the term "the soldier's heart" is, that we are dealing with a soldier; the question of whether we are dealing with his heart or not seems to be lost in a consideration of the general condition of the individual. I am in accord with all that Sir James Mackenzie stated as to the method by which such a condition should be treated. In the hospital to which I am attached we have three wards devoted to soldiers; that is, we have given up nearly half our accommodation to them; and those wards are usually over-full. There have been a considerable number of cases returned incapacitated on account of their hearts, cases—however, in which, on careful examination, one can find nothing amiss with its size, rhythm, or mechanical structure, but with a statement of incapacity to undertake those exertions which were formerly undergone without discomfort. Therefore some factor has been introduced which renders these men incapable of their duties as soldiers. I have found these cases among young recruits only a few years into their teens, and also, usually, in fine-fibred individuals of more or less neurotic type. That it is a valid incapacitation which they evince is undoubted; but I think, a few years ago, instead of the heart being regarded as specially at fault, it would have been the nervous system which would have been chiefly blamed. The neurasthenic, the debilitated and depressed person, would have taken the place of the soldier with a condition of special cardiac over-irritability. The heart itself in such cases shows, as a rule, absolutely no evidence whatever, clinically or otherwise, of physiological disturbance. The men have their flushes and blushes, their accelerations, their palpitation, their dilated pupils, and disturbance on exertion, attributable to the nervous system; but, as far as their hearts are concerned, I have failed to find in them any evidence of exclusively cardiac disorder. I am not in a position to assert that there is not some cryptic organism, or toxæmia, active in the cardiac muscle. Such an agent may be assumed or suggested, but has not been demonstrated.

With regard to the treatment of the condition, there is no doubt the opener has shown the only rational line to be followed—rest, followed by greater freedom of movement and mental change. As to prognosis in these cases, the utmost caution is desirable. The man who breaks down under the circumstances which have been described is one who has a certain instability of the heart inherent, which would probably, under any circumstances of stress, tend to incapacitate him. As regards microbic infection, however, one sees men who have been drained by diarrhoea at the Dardenelles, acutely ill in hospital for a considerable period, with a running temperature and septic conditions, but whose hearts are innocent of obvious damage. On the other hand, we find men who have had no serious illness of any kind coming home incapacitated and neurasthenic; and the probability is that the future utility of such as soldiers is very uncertain.

Dr. O. LEYTON.

I wish to draw attention to one method of investigation and only one, not because the other methods already referred to are not of great value, but because so far it has apparently escaped the notice of the members who have taken part in the discussion. Adopting the definition of "soldier's heart," which has been given by Sir James Mackenzie, I believe that the condition is not due to a single cause, but that the symptoms may arise from disease of the heart or disease of the vaso-motor system. The question presents itself as to whether it is possible to differentiate between these two diseases, and if it is possible, by what method. When a heart of a normal individual is auscultated through a stethoscope which permits the accurate estimation of the ratio of intensities of the first and second sound, it is found that the first sound at the apex is of double the intensity of the second sound at the base. Stethoscopes have been devised by Oertel and by Bock for the purpose of making this observation. The principles upon which the two instruments work are different: the former depends upon the size of a window through which sound may escape, whilst the latter depends upon the size of the hole through which the sound must pass on the way from the chest to the ears of the observer. The two instruments give identical results. Disease of the myocardium leads to an alteration in the ratio of the heart sounds, and occasionally during an attack of

diphtheria, when the toxin has caused an alteration in the myocardium, a reversal of the ratio may be observed, and, as recovery occurs, a return to the normal ratio is usually noted. Observations of this type made upon soldiers suffering from the symptoms of "soldier's heart" showed that in some cases the two sounds of the heart were of equal intensity, and these, I believe, were suffering from myocardial alteration, probably of toxic origin. It was of interest to note that as the ratio approached the normal the symptoms disappeared. In other cases, however, there was no alteration in the ratio of intensity, and in these I am under the impression that the disease was of the vasomotor system. It would be of considerable interest to determine how far observations of this type could be corroborated when applied to very large numbers, and whether, as predicted, nervous shocks, such as those produced by high explosives, lead to disturbances of the vasomotor system, which may last for a considerable period, whilst toxins of bacterial and other origin, such as gases, cause disease of the myocardium.

Dr. FLORENCE A. STONEY.

I wish to point out the very close connexion there is between hyperactivity of the thyroid gland and the tachycardia and breathlessness seen in cases of "irritable heart" and "soldier's heart." This association is a most important one, since the thyroid gland is eminently amenable to treatment. These cases resemble slight cases of Graves's disease. There are many cases of shell shock which show no signs of Graves's disease, and where one can only suppose the thyroid has not been adversely affected, but other cases of shell shock show symptoms of hyperthyroidism definitely dated by the patient as commencing only after one such definite shock or after being buried by an explosion. The tendency to thyroid degeneration is one which runs in families, hyper- and hypo-thyroidism appearing in different members of the family, or in the same individual at different times; that is, the gland in some families shows marked inability to withstand adverse conditions—it is unstable—and if exciting causes such as overwork, over-worry, or pyorrhœa occur, then it degenerates. It is also noteworthy that dental caries, tartar, and pyorrhœa alveolaris are, or have been, present very commonly in these cases, but not universally. The thyroid condition persists after all the teeth are extracted; but in

X-ray work I have found those patients who have had their mouths thoroughly cleansed respond very much more quickly to treatment than those in whom constant oral sepsis persists.

Early cases, such as we are seeing in the base hospitals as a result of the present War, are often unrecognized as to their true nature, above all the connexion of the various groups of symptoms with changes in the thyroid gland, and the importance of this connexion is unrecognized. The result is that they drift on for months and months. They are often looked on as cases of "shell shock," as "neurasthenics," or as making the most of their disability, or are merely given medicinal treatment and are sent out much in the same condition as when they came in, to pass almost at once into some other hospital. One soldier, aged 18, whom I saw lately with quite marked Graves's disease, told me he had been in various hospitals or on furlough ever since he had been rendered unconscious for two hours by a high explosive shell last July, and that he was no better than when sent back to England after this explosion; so that all these weeks of treatment in various hospitals have failed to cure him, or even make him appreciably better; and now at last he has come to us and X-ray treatment has been started. He was seven weeks in one hospital, seven weeks in a second hospital, then he had one week's furlough, five days of which he spent in bed, as he felt so ill. After that he was received into Fulham Military Hospital at the end of November. On admission he was very nervous, had general tremor, his pulse was 120 when he stood, and he had slight fullness of the thyroid. After only two X-ray treatments he is already distinctly more cheerful and less nervous, and his pulse is 100 when he stands; he has less throbbing, less tremor, and he says that he now "feels better than with any other treatment he has had."

That X-rays, when properly and vigorously applied, are a specific for exophthalmic goitre I am quite convinced. I have treated this condition with X-rays during the last seven years, and the results are most cheering. Some of my old cases come back to me for a couple of X-ray exposures when they feel run down and in need of a tonic. I have seen several cases labelled debility or neurasthenia which were really slight cases of hyperthyroidism, which did not respond to ordinary medical treatment, but got rapidly well when their thyroid glands had undergone only the slightest reduction in size.

One may speak of Graves's disease or hyperthyroidism which is either (a) primary, or (b) secondary to, a pre-existent bronchocele.

I have now treated over 100 primary and secondary cases.¹ Most of the cases in the present War are primary, no goitre having been apparent before enlistment, and since neither exophthalmos nor goitre need be present in sufferers from this peculiar degeneration of the thyroid, it is better to describe these cases as "hyperthyroidism," and to divide them into groups according to predominant symptoms.

Group I.—Tachycardia. The most numerous group. These cases come in for breathlessness or fainting, often quite definitely associated with the effects of a shell which has burst in their near neighbourhood, or shortly after they were buried by an explosion; and they say quite definitely they were all right till that sudden great strain. Some have been rendered unconscious by the shell, others have not had such a severe shock; some few have found the strain of training too great, and have been greatly exhausted by the long marches or other heavy physical work. They suffer from giddiness and weakness. The state of the heart is most characteristic. The frequency may be normal, or only slightly exceeding normal when lying down, but on sitting or standing up the rate goes up to 90 to 100 or over, only to quiet down again by several beats on resuming the recumbent posture. The heart is often dilated. A skiagram shows dilatation of the right auricle and rounding of the ventricles. The action is tumultuous. The first sound is short and wanting in tone; this is its main characteristic, the heart may be irregular in force and rhythm, showing poverty of cardiac action. Any murmurs present, though often attributed to V.D.H. (valvular disease), are hæmic, or the result of incompetence due to over-distension, and disappear when the heart quietens down, unless there has been previous endocarditis from some other cause, such as rheumatic fever. The pulse is markedly collapsed between the beats, and the blood-pressure is raised. There is no œdema of the legs till late in the disease, when the heart is failing. Such heart failure, with œdema, is seen in civil practice where the heart muscle has been overstrained for years and is consequently degenerated, but is not seen in these slight early cases, the product of this present War. These patients feel very weak, they suffer with dyspnœa, præcordial pain, palpitation, giddiness and fainting. Even when after treatment the heart has resumed its normal beat, the skiagram shows a rounded condition of the whole organ, which persists for some time.

¹ See paper by myself in the *Lancet*, August 10, 1912; and forty-eight cases in the *Brit. Med. Journ.*, August 31, 1912.

Group II manifests marked perspiration, also tremor, which becomes more marked on the least effort or excitement—the tremor is a fine movement of the hand or may be general—or it may show itself in larger jerky twitchings of the muscles.

Group III exhibits mainly nerve symptoms. These patients tell you "their nerves have gone to bits," and they are put down as cases of shell shock; but besides the effect that shell shock seems to exercise on the higher cerebral centres, it may definitely upset the thyroid gland. These patients are "jumpy" and twitch, they have bad dreams, they sleep badly, the pulse becomes unduly rapid on the least excitement, they dread everything. The doctor tells you they are neurasthenics; but what does this word mean, and what is the underlying physical condition? Is it not the same thing as hyperthyroidism, whether due to loss of nervous control, nervous exhaustion, or intestinal toxin, or other cause?

Group IV shows enlargement of the thyroid as the first sign. This is soft and bilateral in early cases, and may or may not have been noticed by the patient before admission. The swelling is only unilateral where there has been previous benign bronchocele. This enlargement may be so slight as only to be evident when the patient puts his head well back so as to stretch the sternomastoids, and then the fullness becomes evident.

Group V shows eye symptoms and is less common. Exophthalmos may be the first thing noticed, and when tired their eyes *feel* large. The absence of wrinkles on the forehead and von Graefe's sign of jerking lids are more common signs. By the time the man reaches the base hospital two or more groups of symptoms are usually present, and common combinations are: tremors, nerves shaky, and skin moist; or breathlessness, tachycardia, weakness and sweating; or nervous twitchings, no wrinkles in the forehead—in fact, the symptoms of the above five groups may be combined in any proportion. These patients also show some general conditions: they lose weight, feel "good for nothing," they are despondent and irritable, worried by small causes. Nearly all are constipated, though the bowels may be relieved daily, and most of their mouths exhibit various degrees of pyorrhœa and of dental caries.

Having correctly diagnosed the condition, the next thing is to cure it. Of the various methods of treatment I shall only deal with *X-ray treatment*. While this does not pretend to deal with the primary

cause of the disease, it usually cures the patient. By its means we can atrophy the thyroid gland to any desired extent, by vigorous and filtered doses. The advantages of this treatment are: (1) There is no shock or danger to life, as in operation; (2) the gland can be reduced until the secretion becomes normal; (3) there is no pain or unpleasantness. The patient merely lies on a couch and the X-rays are applied to each side of the neck over the gland; the patient does not feel anything, and sometimes goes to sleep during the treatment. The only disadvantage of this treatment is that it requires several sittings spread over a couple of months or more according to the severity of the case.

In hospital the soldiers are ideally placed to get well. They are freed from all worry, well fed and cared for, and can take rest to allow the heart to recover tone. One serious source of delay is that it takes on an average six weeks to two months to get their mouths cleansed, which is a great, almost an essential, aid in treatment. Other points needing attention consist either in thorough abdominal massage or daily aperients to overcome the intestinal stasis, of which the patient does not complain, but which is always present. Rest is useful in restoring the weakened heart, but I do not keep my patients constantly in bed except when the cases are very acute; many of them have had months of bed without result before coming to me, and are thoroughly weary of it.

I apply the X-rays as vigorously as is compatible with safety to the skin. At first I did not filter, and the patients had much mild dermatitis; now I give much bigger doses, filtered—one Sabouraud dose through 1 mm. or 2 mm. of aluminium as a filter, to each side of the thyroid weekly; this requires about twenty minutes. The results are in some cases slow, in others astonishing. A languid, tired, irritable, "jumpy" patient week by week gains in mental quietude, becomes better tempered, more placid, and feels better and stronger. Sleep, digestion and pulse all improve, and he gains weight. The heart is one of the earliest organs to respond: it first becomes regular, then slows down and resumes its normal size while regaining tone in the first sound. The throbbing in the carotids disappears and there is a strong, full, regular pulse, steady and quiet even in the upright position, though for some weeks longer any excitement or emotion raises the rate.

This treatment requires much patience and attention to detail on the part of both doctor and patient, but the good results obtained are quite worth the time and trouble spent, and if more widely used would bring many back to health who now drag on existence in miserable

weakness. The difficulty lies in the time required, and in the patients sometimes feeling so much better that they are discharged before being fully cured. The main fact I wish to lay stress on is that all the symptoms are suggestive of over-secretion of the thyroid gland, and, when so caused, *can be cured* by partial atrophy of the gland. If seen early they can be cured quickly, if left for months they take longer; but they can be cured as certainly as an abscess is cured by opening and draining it.

The following are notes of four cases at Fulham Military Hospital:—

Case I.—E. C., private, aged 22. Perfectly well till he enlisted, June 6, 1915. He felt greatly exhausted while training, finding the long marches especially trying. On July 23, 1915, he was sent to Fulham Military Hospital, suffering from breathlessness and tachycardia. He was kept entirely in bed, but at the end of six weeks there was no improvement in his symptoms. He was intensely nervous, weak, and pale. A skiagram showed a rounded, tumultuous heart. When I saw him I diagnosed hyperthyroidism of the nervous and cardiac types. On September 4 I began X-ray treatment of the thyroid. I did not keep him wholly in bed. He showed slight fullness of his neck, there was no pyorrhœa, he was very breathless, his pulse recumbent was 136, and he was so scared and nervous I could hardly get him to lie quiet on the couch for the first treatment, though later he came willingly. Next week he had his second treatment; still breathless, pulse 104 when recumbent. On October 18, after seven treatments, he was sent on furlough for a month, therefore the X-ray treatment had to stop. His neck was $\frac{3}{4}$ in. smaller, there was no dyspnœa and no tachycardia. He felt strong, and was able to be up all day and was very much quieter in manner. All this was accomplished in six weeks. When seen one month later he was back at his depôt. He told me he was so well he could not get his discharge from the Army. He was slightly nervous in manner, his pulse when standing was 72, and all other symptoms were completely gone; his weight had gone up and he felt strong and well. He went out rather too soon and runs some risk of relapse in consequence.

Case II.—H., private, aged 25. In France November, 1914, to April, 1915; through Ypres and Neuve Chapelle; sent home in April, 1915, for rheumatism, bronchitis, and frost-bite. On September 2, 1915, entered Fulham Military Hospital. Said to be neurasthenic. Weight 8 st. 10 lb. He perspires, has headache, eyes misty, vision normal; he is losing weight; his collars have felt tight of late. An X-ray examination showed the rib cartilages ossified, and the heart small, rounded, and tumultuous. October 18: The note of Dr. Wilson is "high blood-pressure, marked vasomotor symptoms, no gross cardiac lesion, nervous, toxæmia." October 25: His

doctor (Dr. Dainty) and I agreed that his symptoms were really due to hyperthyroidism, so I began the X-ray treatment. He then had fine tremor, perspiration, and pyorrhœa; the pulse was irregular in force and rhythm, 92 recumbent; he had slight goitre. November 11: Third treatment. Pulse regular, 76 when recumbent; pyorrhœa still present, goitre less, no tremor, no perspiration; feels better. By December 16 he had had seven treatments, but further applications were delayed by dermatitis. January 8, 1916: No further treatment; the dermatitis has disappeared. He has improved very markedly since the treatment began; he is no longer depressed, and feels strong, "better than before Neuve Chapelle." He weighs 9 st. 9 lb., pulse is regular, 80 standing. Headaches, tremor and perspiration have all disappeared, and he is able to run without getting unduly out of breath; the goitre has disappeared, his neck measures 1 in. less than when X-rays were begun, two and a half months ago. This case had responded very well to treatment; he has now applied for a commission.

Case III.—W., private, aged 31. In France, November, 1914, to May, 1915. He was buried by the explosion of a shell. Complained of sore feet and excessive perspiration in April, 1915. Admitted to Fulham Military Hospital in June, 1915. He was sent to me for the treatment of excessive perspiration, chiefly of his hands and feet. Local treatment did not succeed, and I noticed his pulse varied from 68 to 100, while he had distinct fine tremor. I therefore applied X-rays over his thyroid gland. After five treatments the tremor was gone and perspiration much diminished. The pulse was 80 standing. He was discharged. He would have been the better for a few more applications.

Case IV.—M., private, aged 21. In France October to December, 1914—home wounded. In France March to October, 1915. Admitted to Fulham Military Hospital, October 9, 1915, and palpitation and disordered action of the heart were diagnosed. He had been suffering with palpitation four months, gradually getting worse, shortness of breath, dizziness, and headache. October 26: Præcordial pain; beats hurried, pulse 96, fullness of neck suggesting enlargement of thyroid. First treatment (condition as above): No heart murmurs; pulse 104, regular; goitre large, soft, uniform; the neck measures 14 in. Tremor and perspiration both present, palpitation frequent. November 9: Third treatment; feels better, has felt heart quieter; pulse 100. December 16: Eighth treatment; only one attack of palpitation last week; goitre smaller, 13½ in.; tremor absent, pulse 88 when standing. His doctor considers him a great deal better. December 29: Tenth treatment; palpitation occasional, perspiration and tremor gone; pulse 92 standing; goitre, 13½ in., has almost disappeared; no dyspnœa, no dizziness, no headache; occasional pain in the chest still; is up and about all day. The septic teeth and digestive trouble have delayed the progress of this case.

So far I have had twenty-one soldiers with tachycardia, excessive perspiration, or shell shock under my care. Some have been given thyroid (which made them worse) or parathyroid (which had no effect), and have also had months of rest and heart tonics. Most of these cases are still under X-ray treatment, and it is too soon to report on them.

I am indebted for these cases to the kindness of the other medical officers of the Fulham Military Hospital who allow me to treat their cases, and especially am I indebted to our Commanding Officer, Major Parsons, M.D., for his constant help and interest.

Dr. T. R. BRADSHAW (Major, R.A.M.C., T.).

I did not intend, when I came to the meeting, to say anything myself. I have practically been sent here by the First Western General Hospital to gain information as to how to deal with these cases, which are of such special interest to those of us who are on the staff of the base hospitals where these patients are, and where we have, very often, finally to dispose of them and to decide what is to be their future as regards the Army. Sir James Mackenzie has done more than any living man—and perhaps more than any man who has ever lived—to encourage us in dealing with heart cases. I think there is no more hopeful book than his treatise on the heart, and I am certain he has been the means of restoring to activity countless sufferers from—perhaps I might rather say proprietors of—murmurs, people who in former days would have been condemned to a life of invalidism, but who, owing to his sound teaching, have been found fit to take their part in the battle of life. I know of such cases myself, and of the immense benefit and happiness accruing, not only to the patient, but also to the medical man, from the teaching of the opener of the debate.

There is one point I have hardly heard mentioned, but which has a considerable bearing on this question; and I am looking at it at present not so much from the point of view of pathology, but as a practical matter. It is this: I have frequently had men from the Front sent to my ward as patients, and on being asked what they have come for have replied, "V.D.H., sir." They may bring a slip of paper bearing the words "Valvular disease of the heart"; yet not one of them has had a murmur. Either valvular disease of the heart has been diagnosed on

insufficient grounds or it is a condition which passes off. I leave you to decide. I have not been to the Front, but the refinements of diagnosis which we try to carry out at the base hospitals are obviously hardly possible there. But, as Sir James Mackenzie hinted, it is unfortunate that when there is a doubt about a case, it should be labelled valvular disease, because when a man has once got those letters opposite his name they are, like a bad character, difficult to remove. We must be prepared to consider these cases on their merits, without attaching too much importance to the fact that the man is stated to have valvular disease.

The practical point I want to take home in my report to my Colonel is the reply to the question: What is the prospect of our being able to do sufficient for these men to enable them again to become efficient soldiers? If there is no reasonable prospect of that, I think we are best doing our duty to the State and to the men themselves by invaliding them out of the Army as soon as possible. I should be glad if Sir James Mackenzie would, in his reply, give us some lead on this point. Short of becoming efficient soldiers for the Front, are they fit for home service? We have heard the treatment laid down and discussed, and I think special provision will have to be made for that; certainly cheerful exercises seem the most promising. I cannot, from my own experience, endorse the enthusiastic report of X-ray treatment in exophthalmic goitre which we have heard from one speaker. No doubt in some cases it does good, but there are many in which it does no good.

DR. BEZLY THORNE.

I shall not enlarge on the clinical picture, which has already been graphically and precisely drawn by preceding speakers, of a condition which I have already more than once defined, and have ventured to call "cardio-vascular atony." I have laid emphasis on the vascular element of the syndrome because its characteristic phenomena are largely attributable to deficient action of the muscular and elastic constituents of the vessels, whether as the result of structural deterioration or of defective innervation, or of both. It is that condition which largely accounts for the abnormal postural variations of pulse frequency, while myocardial debility is responsible for murmurs, likewise influenced by posture, which occur quite independently of valvular lesion. I maintain

that cardio-vascular atony is frequently met with in children and adolescents, and I submit that that fact, taken in conjunction with the ratio of its incidence in men returning from the Front, suggests that they have been previously subject to it in a minor degree, and have suffered an acute exacerbation as a result of the strain to which they have been subjected. Those who have been subjected to the influences of high explosives exhibit, in a degree out of proportion to the other symptoms, fibrillar twitchings of the extended fingers and an occasional jerk of the arm. Such conditions coexist invariably with a moderate dilatation of the heart, seldom carrying the apex beyond the line of the nipple, which might easily be overlooked or regarded as a negligible quantity. As a matter of fact it is, however, the dominant factor of a syndrome; the primary and causative condition is a toxæmia, more often than not, of the alimentary order.

Some speakers have appeared to despair of the efficacy of drugs in these subjects and of their ever being able to return to active service or active life, while others have rightly emphasized the importance of an early return to moderate exercise. I am happy to say that each case that has been under my care has been restored to health and activity in a period of two months or a little more. Only those whose pulse has risen above 120 have been kept recumbent, and that generally for not more than a week. The dilatation has been at once attacked by the so-called Nauheim treatment, and has yielded rapidly. The fibrillar twitchings and arm-jerks have subsided in two or three weeks; the coldness of the extremities, the mental depression and irritability giving way *pari passu*.

I now cite two illustrative cases: one that of a young combatant officer who was invalided home for seven months, undergoing treatment by rest and heart and nerve tonics without making material progress; the other a captain, R.A.M.C., who was sent home as a "lying" patient, as it had not been considered advisable that he should maintain the erect posture. Both were in the retreat from Mons, and the first-mentioned subsequently underwent a trying experience in the trenches. Both returned to full vigour in less than two months under Nauheim treatment. I earnestly recommend that those who have charge of such cases should give that treatment a fair and persevering trial.

Sir JAMES MACKENZIE, M.D., F.R.S. (in reply).

I do not think I need detain the meeting with further remarks, because we are still carrying on an investigation, and I am hoping that we shall get from the Government a heart hospital which can be devoted to these cases; and that we shall get a number of men specially told off, and who are specially qualified to work out all the problems, and who will investigate this subject thoroughly. I am hopeful that we have the lines on which we can pursue the matter with profit, and I trust that we shall be able to come before you with much more definite information than we could submit to you to-day.

Therapeutical and Pharmacological Section.

President—W. HALE WHITE, Lieutenant-Colonel R.A.M.C.(T.F.).

New Treatment of Tuberculosis.

DR. GEORGES TH. PANOUPOULOS, of Athens, has sent to the Section the following note of what he believes to be a new treatment for tuberculosis, in the hope that members will try it and communicate their results to him. Members who are interested in the subject may communicate direct with : Dr. Georges Th. Panoupoulos, Chateaubriand 12, Athens.

The procedure consists in the destruction of Koch's bacillus by means of ozone formed in the lungs under the agency of the inspiration of pure oxygen and the simultaneous action of hard X-rays.

The application of my method is as follows :—

Before radiation is started the patient is made to breathe pure oxygen by means of an apparatus which is made so that the entry of atmospheric air into the lungs is prevented. This is of very great importance, because the presence of the large amount of nitrogen contained in the atmospheric air, during the action of the rays, would convert the nitrogen in the lungs into oxide of nitrogen, which would have an injurious action on the tissues. After five minutes' inspiration of the oxygen, the action of the hard X-rays or other radio-active bodies should be commenced, the patient continuing to breathe the oxygen throughout the treatment.

In order to avoid X-ray burns, a filter consisting of an aluminium plate 0.5 mm. to 2 mm. in thickness should be interposed.

The duration of the action of the X-rays, the intensity of which varies from 2½ H. to 8 H. at each sitting, is from ten to twenty minutes.

The X-rays (or other radio-active substances) convert a very small portion of the pure oxygen which fills the lungs into ozone, the bactericidal action of

which is well known. To this very important action of the ozone must be added the combined action of radio-activity and of the oxygen, which insures :—

(a) The increase of the beneficial oxidation of chemical combinations of the blood (hæmospherine, hæmoglobin, &c.), due on the one hand to the presence of a large quantity of oxygen, and on the other to the ionization of the oxygen, the oxidizing and diffusive action of which is increased as a result of the catalytic action of the X-rays.

(b) The influence brought to bear on the tubercles and the bacilli themselves.

I began the application of this method of treatment with the approval of eminent physicians of Athens in the month of March, and the results I have obtained are very encouraging.

As soon as I am in a position to work with two apparatus at once I shall induce the X-rays from two sides, so as to insure a "cross-fire" action of these rays in the lungs. I have every reason to believe that I shall then have increased not only the quantity of formed ozone, but also the activity of the X-rays and of the oxygen.

Therapeutical and Pharmacological Section.

President—W. HALE WHITE, Lieutenant-Colonel R.A.M.C.(T.F.)

(May 16, 1916.)

DISCUSSION ON THE TREATMENT OF DIABETES MELLITUS BY ALIMENTARY REST.

Opened by O. LEYTON, M.D.

ANY man who would open voluntarily a discussion on diabetes mellitus must possess exceptional courage, or exceptional knowledge, or exceptional ignorance. I do not lay claim to any of these, and wish to draw attention to the fact that the discussion is upon a particular form of treatment of the disease, a treatment which has been adopted during the last two years.

In order to discuss the treatment of a disease we need not be acquainted with the cause of the condition, as shown by the fact that quinine cured malaria long before Laveran demonstrated the presence of the plasmodium; mercury and potassium iodide diminished the ravages of syphilis for many years before Schaudinn and Hoffmann found the *Spirochæta pallida*; whilst salicylates, introduced by Mackenzie, ameliorate rheumatic fever, a disease the cause of which is still under discussion. That the discovery of the cure of a disease may precede the discovery of the cause of the disease is an unfortunate fact, for it leads to some optimists devoting their time to empirical observation—observation which is rarely fruitful. But, even if it is granted that we need not know the cause of a disease in order to be able to discuss its treatment, we must have some definition of the disease in order that the speakers who take part in the discussion may talk about the same disorder.

DEFINITION.

There is no satisfactory definition of diabetes mellitus, and therefore, for the purpose of the discussion to-day, with your permission, I shall assume that a patient who, whilst upon normal diet, passes dextrose in the urine in quantities sufficient to be detected by the reduction of Fehling's solution is suffering from diabetes mellitus, provided that he does not present any symptoms of disease of the central nervous system or thyroid gland; this excludes pentosuria, lactosuria, and lævulosuria.

It is true that this definition fails in that, occasionally, the reduction of copper in Fehling's solution would not occur with the amount of sugar present in the urine were the reduction not assisted by glycuronic acid or other reducing substances. The difficulty then is as to what is responsible for the reduction—the minute amount of dextrose or the glycuronic acid? It is scarcely necessary to emphasize the immense difference between the two statements: "who passes dextrose in the urine in a quantity to be detected with Fehling's solution," and "who passes urine which will reduce Fehling's solution." The certainty that the substance causing the reduction is dextrose must be established either by fermentation or by forming an osazone and determining its melting-point.

Perhaps the most convenient definition for the clinician would be the passing of urine containing sufficient sugar to be detected by the fermentation test; but this might exclude a few early slight cases of diabetes mellitus and would include lævulosuria.

Although the above definition is not satisfying, until we know the cause of the disease we cannot expect an accurate definition, especially when we remember that normal urine contains about 0.01 per cent. sugar, whilst in diabetes mellitus there is simply an increase in the amount along with other alterations in metabolism, varying in intensity with the gravity of the condition; nevertheless, the definition is logical, as shown by the experiments of F. M. Allen, who has found that if a small fraction of pancreas be removed from a dog, the animal's metabolism is disturbed and sugar will appear in the urine in larger quantities than normal. On removing a bigger fraction, the dog's metabolism is still further upset, and when only one-tenth of the pancreas is left, all the symptoms of severe diabetes developed.

We must not draw the conclusion from these experiments that diabetes mellitus is due to degeneration of the pancreas. The defini-

tion agrees with a statement of Joslin, which I was fortunate enough to come across after adopting the above, who writes:—

“I consider any patient to have diabetes mellitus, and treat him as such, who has sugar in the urine demonstrable by any common tests. This is a broad definition, but it is safer for the patient than the use of the term glycosuria, which begets indifference.”

If we adopt this definition we may banish the idea that there are two diseases—diabetes mellitus and dietetic glycosuria—the former of grave and the latter of slight importance, and acknowledge that the two conditions are two varieties of the same disease.

It is unwise to divide diseases into many classes when the classification must depend purely upon the time between the onset of the disease and the death of the patient. Although we are including all cases of dextrosuria into the class of diabetes mellitus we must recognize that it is extremely probable that there are several pathological conditions which give rise to the condition. This we must keep before us continuously when considering the effects of treatment, and realize that the results of treatment may assist us in differentiating the causes.

COURSE OF THE DISEASE.

In order to determine whether a treatment is of value we must be well acquainted with the usual course of the disease: the greater the deviation from the average the greater the difficulty in assessing the value of any special form of treatment; and therefore our duties to-day are onerous if we are to arrive at a definite conclusion.

The variability is so great as to make prognosis with the older methods of investigation an impossibility, and even with those at our disposal at the present time, men with the most experience will have the greatest hesitation in making a prophecy.

Many of us have seen isolated cases of the disease in the young recover. In 1910 I saw a youth, aged 17, who had become so thin that his father would not go out with him because, if he happened to meet any acquaintance, he was always accused of starving his son. This youth put on 3 st. in eight months, and is quite well at the present time, an officer in Egypt. Nine years ago a distant relative of mine consulted several eminent physicians, all of whom told her to go home and make her will, because she was passing 6 per cent. dextrose in the urine and suffering from polydipsia and polyuria, along with wasting

and œdema. She decided that as the prognosis given was so depressing she would enjoy the remainder of her life, and eat and drink whatever she fancied. She died a few months ago, but not from diabetes mellitus. In 1909, a youth, aged 18, was sent to me with the history that he had passed urine containing more than 7 per cent. of sugar, had been put upon diabetic diet, and all the sugar had disappeared. He then returned to a normal diet, and was free from sugar for some months. Glycosuria recurred, and on a modified constant diet without any apparent cause this percentage of sugar varied from 0.4 per cent. to 6 per cent.—that is to say, the quantity of sugar he excreted one day was 4 grm. and the next 80 grm. This youth lived for seven years after the date upon which 7 per cent. sugar had been present in his urine.

I am indebted to Mr. Barrett (of Colwyn Bay) for being allowed to refer to a remarkable case of a man suffering from diabetic coma, in whom an injection of sodium carbonate not only led to the usual temporary recovery, but to the patient living more than five years after the attack of diabetic coma. The cause of death was pneumonia.

It is not my intention to detract from the value of treatment in cases of this type, but the memory of a patient whom I saw five years ago makes me realize that occasionally recovery from diabetic coma takes place without any apparent cause. A lady, aged 55, who had suffered from diabetes mellitus for more than four years, was suffering from air-hunger and coma; could not be roused by noise or shaking; there was acetone in her breath. The practitioner and I gave a most grave prognosis, telling the husband that it was extremely unlikely that his wife would rally, and asking him whether he would like us to give an intravenous injection of alkali, because we were of the opinion that it would lead to temporary return of consciousness. He took the view that this would be harrowing to the patient and relatives, and that it would be cruel to awaken her for so short a time. No injection was given. The patient recovered, and I heard quite recently that she was alive.

It is this great variability which has made it so difficult to decide whether any drug or treatment is really beneficial, and has led to all kinds of drugs being advocated as cures. Time prevents my enumerating them in detail, but amongst them we can number uranium salts, calcium iodide, vaccines of various types, and enzymes. No doubt individual cases have improved after the administration of each, but probably it should be written "in spite of the drug" rather than "due to it."

TREATMENT.

For many years the approved diet in diabetes was one containing as little carbohydrate as possible; much protein and fat were pressed upon the unfortunate patient with the hope that they might lead to a gain in weight.

Naunyn found that if a patient were made to fast for a day or two the quantity of sugar fell rapidly, and he believed that by feeding the patient generously for the next day or two he was able to build up the body.

The fast-days were later adopted by von Noorden, who introduced the so-called "green vegetable days." The green vegetables selected contained less than 5 per cent. available carbohydrate and very little else, so that a green vegetable day approached a fast-day.

Guelpa adopted the view that diabetes was due to auto-intoxication, and treated the disease by fasting and purging.

The literature is full of papers recommending modification of Naunyn's and Guelpa's suggestions, but as far as I can find the real advance has been made by F. M. Allen, of the Rockefeller Institute. I cannot do better than quote the actual words used by the author of the treatment which seems to me to be proving the most satisfactory. Dr. Allen writes:—

"When the fasting patient has been free from glycosuria for twenty-four to fifty-eight hours, the next step is to begin feeding very slowly and cautiously. There need not be a fixed programme. It is desirable to individualize the diet to suit the needs of different patients, and various physicians may have personal preferences of their own. The one requirement is that the patient must remain free from both glycosuria and acidosis. Any trace of sugar is the signal for a fast-day, with or without alcohol. The original fast, to clear up the urine in the first place, may be anything from two to ten days, but after that no fast need be longer than one day. The things to be considered in the diet are carbohydrate, protein, fat, and bulk. Frequently the first thing given after the fast is carbohydrate. No distinction is necessary between different forms of starch, but there are advantages in using vegetables, following Joslin's convenient classification on the basis of carbohydrate content. The first day after fasting the only food may be 200 gm. of vegetables of the 5 or 6 per cent. class. This is increased day by day until a trace of glycosuria appears, which is checked by a fast-day. The purpose of such a programme is to learn the carbohydrate tolerance and to clear up the last traces of acidosis. After this carbohydrate period, or sometimes in place of it, protein is given and nothing else. More protein, generally as eggs and meat, is added day by day, until the patient either shows glycosuria or reaches a

safe proteid ration. The purpose here is to learn the protein tolerance, and to cover protein loss as quickly as possible. Fat is somewhat less urgently needed, except in very weak and emaciated patients, and it can be added gradually, as conditions seem to indicate. An element of bulk in the diet is necessary to give the comfortable feeling of fullness and to prevent constipation."

These few words sum up the treatment, but leave much to the individual physician to decide and perhaps to come to some definite decision in each case. It is therefore of interest to note in what way Dr. Joslin, of Boston, has standardized the suggestions in Allen's remarks:—

"*Fasting*.—Fast until sugar-free. Drink water freely, and one cup of tea and one cup of coffee if desired. If sugar persists after two days of fasting add, in divided portions, 300 c.c. clear meat broth.

"*Alcohol*.—If acidosis (diacetic acid) is present, take 0.5 c.c. alcohol per kilogram body-weight daily until acidosis disappears. Alcohol is best given in small doses every three hours.

"*Carbohydrate Tolerance*.—When the twenty-four-hour urine is sugar-free add 150 gm. of 5 per cent. vegetables, and continue to add 5 gm. carbohydrate daily up to 20 gm., and then 5 gm. every other day, passing successively upward through the 5, 10, and 15 per cent. vegetables, 5 and 10 per cent. fruits, potato, and oatmeal to bread, unless sugar appears or the tolerance reaches 3 gm. carbohydrate per kilogram body-weight.

"*Protein Tolerance*.—When the urine has been sugar-free for two days add 20 gm. protein (three eggs), and thereafter 15 gm. protein daily in the form of meat until the patient is receiving 1 gm. protein per kilogram body-weight, or if the carbohydrate tolerance is zero, only $\frac{1}{2}$ gm. per kilogram body-weight. Later, if desired, the protein may be raised to 1.5 gm. per kilogram body-weight.

"*Fat Tolerance*.—While testing the protein tolerance, a small quantity of fat is included in the eggs and meat given. Add no more fat until the protein reaches 1 gm. per kilogram (unless the protein tolerance is below this figure), but then add 25 gm. fat daily until the patient ceases to lose weight or receives not over 40 calories per kilogram body-weight.

"*Reappearance of Sugar*.—The return of sugar demands fasting for twenty-four hours, or until sugar-free. The diet preceding the reappearance of sugar is then resumed except that the carbohydrate should not exceed half the former tolerance until the urine has been sugar-free for two weeks, and it should not then be increased more than 5 gm. per week.

"*Weekly Fast-days*.—Whenever the tolerance is less than 20 gm. carbohydrate, fasting should be practised one day in seven; when the tolerance is between 20 and 50 gm. carbohydrate, 5 per cent. vegetables and one-half the usual quantity of protein and fat are allowed upon the fast-day; when the tolerance is between 50 and 100 gm. carbohydrate the 10 per cent. and

15 per cent. vegetables are added as well. If the tolerance is more than 100 grm. carbohydrate upon the weekly fast-day, the carbohydrate should be halved."

The advantages of a treatment may be investigated from two points of view: that of the patient and that of the adviser. The advantages claimed by Allen are that it is possible to keep a patient's urine free from sugar and free from evidence of acidosis. In addition to this, alterations in metabolism may be demonstrated—alterations which make the patient approach the normal. First, the effect of fasting in a fairly severe diabetic is most marked, and one of the extraordinary things about it is that acidosis diminishes, which is in great contrast to the effect upon a healthy individual: when a normal individual is made to fast, acetone appears in the urine.

If the clinician were satisfied only with a treatment which cures the patient he would be one of the most dissatisfied of individuals upon this earth, for the treatments which really cure may be counted upon the fingers, and consist perhaps of quinine, arseno-benzol, and iodides: quinine in malaria, arseno-benzol in disease due to certain treponemata, and iodides in sporotrichosis. In many cases the clinician must be content if he can prolong the life of his patient and keep his patient free from pain and discomfort. Medical science cannot supply new organs, and often damaged organs will not regenerate.

The majority of cases of diabetes mellitus are due to an alteration in the functions of the pancreas. Occasionally this alteration is temporary, and recovery occurs; usually it is permanent, and all that can be done is to arrange a diet so that the functions of the damaged organ are not overstrained. Therefore, if any treatment of diabetes mellitus prolongs the life of the patient, relieves thirst and hunger, prevents complications such as abscesses and neuritis, that treatment must be considered satisfactory.

The modern clinician will be able to determine the efficiency of the treatment by observing the alteration in metabolism:—

- (1) The sugar in the urine.
- (2) The sugar in the blood.
- (3) The respiratory quotient.
- (4) The alveolar air.
- (5) The alteration in the quantities of β -oxybutyric acid and other acids excreted.
- (6) The body-weight.

(1) *The Sugar in the Urine.*

That fasting leads to a rapid fall in the quantity of sugar in the urine has been established beyond doubt; as an illustration, I refer to a case of a man, aged 55, who had suffered from diabetes for three years:—

Date	Volume of urine	Specific gravity	Percentage of sugar	Sugar, grammes
April 29, 1914 ...	3,360 ...	1030 ...	4.3 ...	144.0
" 30, " ...	2,610 ...	1032 ...	4.5 ...	117.0
May 1, " ...	720 ...	1032 ...	1.6 ...	11.5
" 2, " ...	540 ...	1029 ...	0.1 ...	0.5

(2) *Percentage of Sugar in the Blood.*

A discussion upon the real nature of the substances which we estimate as sugar in the blood would be of immense interest, but must be postponed to a more suitable time, and for convenience to-day I shall talk of these substances as sugar.

The estimation of sugar in the blood by the method suggested by Lewis and Benedict is speedy, and a mental recreation, so that in the near future we shall possess many more data upon this point. The average percentage of sugar in normal blood is 0.1 per cent. We must realize that so far no relation has been discovered between the percentage of sugar in the blood and that in the urine. Diabetes mellitus and hyperglycæmia are not synonyms. The urine may contain quite a large percentage of sugar, whilst the blood may have less than 0.1 per cent.; on the other hand, the blood may contain 0.3 per cent. of sugar and the urine may contain too little to be detected by Fehling's test.

Date	Volume of urine	Percentage of sugar in urine	Percentage of sugar in blood
May 5, 1916 ...	2,940 ...	9.1 ...	0.277
" 11, " ...	630 ...	0.5 ...	0.23
" 15, " ...	720 ...	0 ...	0.19

This is better shown in a case recorded by Joslin:—

Date	Volume of urine	Percentage of sugar in urine	Percentage of sugar in blood
April 15, 1914 ...	1,540 ...	2.52 ...	0.26
" 16, " ...	1,420 ...	3.15 ...	0.43
" 17, " ...	900 ...	0.19 ...	0.26
" 18, " ...	680 ...	1.1 ...	0.33
" 19, " ...	580 ...	0.5 ...	0.29
" 21, " ...	540 ...	0 ...	0.23
" 24, " ...	570 ...	0 ...	0.31
" 27, " ...	640 ...	0 ...	0.31
May 1, " ...	945 ...	0 ...	0.28

It is true that not uncommonly hyperglycæmia and glycosuria go hand in hand, and when the glycosuria ceases it is found that the sugar in the blood has fallen to 0·12 per cent.

Date	Volume of urine	Specific gravity	Percentage of sugar in urine	Percentage of sugar in blood
April 28, 1916 ...	2,880 ...	1039 ...	5·0 ...	0·24
" 29, " ...	1,680 ...	1027 ...	3·0 ...	—
" 30, " ...	1,800 ...	1027 ...	3·0 ...	—
May 1, " ...	1,200 ...	1014 ...	1·1 ...	0·14
" 2, " ...	1,320 ...	1013 ...	0 ...	—
" 3, " ...	1,200 ...	1012 ...	0 ...	0·12
" 4, " ...	1,680 ...	1014 ...	0 ...	—

Three conditions, possibly three diseases, must be recognized: glycosuria without hyperglycæmia, glycosuria with hyperglycæmia, and hyperglycæmia without glycosuria. The explanation of these phenomena may appear to be that the methods at our disposal at the present day do not differentiate between two conditions: (1) dextrose in the blood, (2) substances which are changed to dextrose and estimated as dextrose. Attractive as this explanation may appear to be, there are cogent arguments against it, for experiment has shown that the introduction of pure dextrose into the blood-stream may fail to cause any glycosuria; and again cases of glycosuria have been recorded which have started with hypoglycæmia and developed into hyperglycæmia; upon this point Dr. Langdon Brown will give us some illuminating statements. Some assume that the complications, such as abscesses, neuritis and cataract, are directly due to the increased quantity of sugar in the blood. Assertions that there can be no doubt about this must not be accepted as proof. Repeated intravenous injections of solutions of dextrose into various animals have failed to produce these conditions, and the possibility of some other change in metabolism being responsible must be considered.

The more we think over these observations, the more difficult it is to explain them. We see that in certain cases the kidney, through which blood containing 0·3 per cent. sugar is passing, turns out urine free from sugar; whilst in another case, a kidney supplied with blood containing no more than 0·1 per cent. excretes considerable quantities of sugar. It is true that injections of phloridzin produce somewhat similar effects, and it is within the realms of possibility that a phloridzin-like body is prepared by abnormal metabolism in cases of severe diabetes, and this substance ceases to be made on withholding food from the individual. But we shall see after discussing the effect of alimentary

rest upon the respiratory quotient that this explanation fails to satisfy the data before us. We must therefore hesitate in attaching more importance to the percentage of sugar in the blood than that in the urine.

(3) *Respiratory Quotient.*

When the organism is unable to utilize the carbohydrate supplied to it, the respiratory quotient falls to the region of 0.7; and rises as the capacity for oxydizing carbohydrates or for utilizing the carbohydrate radical of protein increases. In several cases it has been found that by treatment the respiratory quotient has risen from 0.72 to 0.76, demonstrating a marked improvement in being able to utilize carbohydrate.

Eugene Du Bois found that in a severe case of diabetes mellitus the metabolism was 8 per cent. above normal, and that the patient was excreting all, or nearly all, of the sugar formed from protein and burning practically no sugar. In a nine days' fast, glycosuria ceased, the total metabolism fell to about 20 per cent. below normal and the respiratory quotient showed that sugar formed from protein was being burnt.

This observation tempts speculation: Is it that alimentary rest allows the pancreas to stop secreting pancreatic juice, and that this permits it to carry out its internal secreting process in a more complete manner?

(4) *The Alveolar Air.*

Examination of the alveolar air leads one to the same conclusion as the respiratory quotient, and is a more rapid method and better adapted for use in the wards.

(5) *The Production of β -oxybutyric and other Acids.*

The most rapid method of arriving at some idea of the quantity of these acids excreted is by the estimation of the ammonia in the urine; this estimation, if it is allowed to include amino acids by the formic aldehyde method, is extremely rapid; but one must remember that Cammidge has shown that the estimation of amino acids is of very great value, and the greater the quantity appearing in the urine, the worse the prognosis. A rapid fall in the quantity of ammonia appearing in the urine the worse the prognosis. A rapid fall in the quantity of ammonia appearing in the urine upon starvation shows that the production of these acids must diminish rapidly.

(6) *Body-weight.*

One would expect a patient to lose weight fairly rapidly even if kept in bed and supplied with a certain amount of alcohol. The loss of weight does not cause any great inconvenience, and usually within a fortnight of the beginning of the treatment a diet has been arrived at which causes loss of weight to be extremely slight, and after a further period weight becomes stationary, or it may even increase a few ounces during the week.

The difficulty in carrying out the treatment depends greatly upon the surroundings of the patient. Before we have had experience and have satisfied ourselves that the benefits accruing are considerable, we do not feel justified in explaining to the patient the gravity of his condition, for it is only when one is certain that one can do good that it is permissible to arouse alarm in the patient. Without explaining what may happen if treatment be not adopted, the average patient refuses to undergo the discomfort of several days' fast and then a restricted diet for some weeks; but my impression is that by having some number of patients undergoing a similar course of treatment in the same ward any tendency to rebellion in one of them is neutralized by the others. One patient who is taking a fair diet after having been through the treatment, and having lost his thirst and polyuria, induces those who are at the beginning of the treatment to see it through.

In private, the success of the treatment depends to a large extent upon the confidence which can be instilled into the patient. Sympathizing relatives should be prevented from seeing the patient and expressing their foolish views on the question of food. In my experience, the administration of thrice-cooked vegetables seems to possess a greater use than many have attributed to it, and rather than give a few ounces of vegetables which have been cooked once I prefer giving two or three times the quantity which have been cooked three times. These satisfy the hunger temporarily.

The treatment has its limits, and I venture to prophesy that we shall find that certain forms of diabetes mellitus may progress best when passing a certain amount of sugar in the urine.

The diet of a typical case is given in detail. After the fast and the urine had become free from sugar the diet allowed was: For breakfast—cocoa, with 2 oz. of boiled celery. For dinner—2 oz. of greens. For tea—a cup of tea. For supper—1 oz. of seakale.

On February 26 (a week later): For breakfast—cocoa, and 4 oz. of

mushrooms. At mid-day—an egg. For dinner—4 oz. of spinach and 3 oz. of cooked meat. Tea in the afternoon. For supper—4 oz. of seakale and an egg.

On March 1: For breakfast—cocoa, egg, and 4 oz. of mushrooms. For dinner—4 oz. of artichokes and 4 oz. of meat. For tea—an egg. For supper—4 oz. of turnips, 2 oz. of fish, and an apple.

On March 10: For breakfast—cocoa, an egg, 1 oz. of bacon, and 1 oz. of cooked oatmeal. For dinner—4 oz. of cooked macaroni and 2 oz. meat. For tea—an egg. For supper—4 oz. of potato, 2 oz. of cabbage, 2 oz. of fish, and 1 oz. of nuts.

On March 17: For breakfast—cocoa, an egg, 1 oz. of bacon, and 2 oz. of cooked oatmeal. For dinner—4 oz. of cooked meat and 8 oz. of 5 per cent. vegetables. For tea—an egg. For supper—cup of clear soup, 4 oz. of fish or meat, 4 oz. of 10 per cent. vegetables, and an orange.

On March 24: For breakfast—cocoa, two rashers of bacon, 2 oz. of dry oatmeal, and 1 oz. of cream. For dinner—2 oz. of cooked meat, 4 oz. of 5 per cent. vegetables, 4 oz. of mushrooms, and 1 oz. of butter. For tea—an egg in clear soup. For supper—Clear soup, 2 oz. of fish or meat, 2 oz. of potato, 2 oz. of 5 per cent. vegetables, $\frac{1}{2}$ oz. of butter, and an orange.

On April 9: For breakfast—cocoa, two rashers of bacon, an egg, 1 oz. of dry oatmeal (cooked), and 1 oz. of cream. For dinner—3 oz. of cooked meat, 4 oz. of 5 per cent. vegetables, 2 oz. of 10 per cent. vegetables, and 1 oz. butter. For tea—an egg and $\frac{1}{2}$ oz. butter. For supper—clear soup, 2 oz. fish or meat, 2 oz. of potato or macaroni, 2 oz. of 5 per cent. vegetables, 1 oz. of butter, and grape-fruit.

On April 16: For breakfast—cocoa, two rashers of bacon, an egg, 1 oz. of dry oatmeal (cooked), 1 oz. of cream, 1 oz. of white bread, and $\frac{1}{2}$ oz. butter. For dinner—2 oz. of cooked meat, 4 oz. of 5 per cent. vegetables, 2 oz. of 10 per cent. vegetables, and 1 oz. of butter. For tea— $\frac{1}{2}$ oz. of cream, $\frac{1}{2}$ oz. of white bread, and $\frac{1}{4}$ oz. of butter. For supper—clear soup, 2 oz. of meat, 2 oz. of potato or macaroni, 2 oz. of 5 per cent. vegetables, 1 oz. of butter, and grape-fruit.

On April 29: For breakfast—cocoa, two rashers of bacon, an egg, 1 oz. of dry oatmeal (cooked), 1 oz. of white bread, and $\frac{1}{4}$ oz. of butter. For dinner—2 oz. of cooked meat, 2 oz. of 10 per cent. vegetables, 1 oz. of butter, and $\frac{1}{2}$ oz. of rice (cooked as pudding). For tea—tea with cream, 1 oz. of bread, $\frac{1}{4}$ oz. of butter, and $\frac{1}{2}$ oz. of sponge cake. For supper—clear soup, 2 oz. of cooked meat, 2 oz. of potato or macaroni, 2 oz. of 5 per cent. vegetables, 1 oz. of butter, $\frac{1}{2}$ oz. of pudding, and grape-fruit.

The diet at present consists of 60 grm. protein, 165 grm. fat, and 97 grm. carbohydrate, which gives energy of 2,113 calories.

On one occasion, when the quantity of rice was increased too rapidly, the patient had sugar in the urine. She fasted for one day and then was free from sugar.

You will note that so-called diabetic preparations make no appearance in the list. Since the maximum amount of protein allowed is 1 grm. per kilogram weight, one can understand that this is taken as meat and eggs, and the type of bread wanted is one containing much cellulose and a little starch. I must add that this is a case of a girl, aged 22, who had been under treatment for two years and had been passing 3 per cent. of sugar while upon a so-called antidiabetic diet.

Most of us have seen some number of cases of diabetes mellitus which have developed tuberculosis, and it is remarkable that, when they develop tuberculosis, sugar frequently disappears from the urine. The case I have just referred to I have examined in considerable detail, and have failed to find any evidence of tuberculosis. I have quoted this case simply as an instance; there are quite a number which have come under my observation that are running a similar course.

No one will claim that the treatment is a cure, or even the most perfect which can be devised. It is my impression that it is a great advance upon any in general use, and that those which regulate the quantities and ratios of the constituents of a diet by frequent analyses of the urine to determine the quantities of urea, ammonia, acetone, aceto-acetic acid, oxybutyric acid, and total nitrogen, combined with an initial fast, approach perfection.

Allen has shown that the tolerance for carbohydrate in many cases of diabetes mellitus depends upon the amount of exercise taken; the greater the exercise the greater the tolerance. The rules supplied by Allen will allow this increased tolerance to be utilized by the ordinary worker. Therefore, as a practical method, it may be of service to the diabetics amongst the labouring classes, whilst the more complex methods have a very limited application.

So far only one case has proved a failure—a young sailor who developed diabetes mellitus shortly after the Battle of the Falkland Islands. During that time he was looking after the electric plant in one of the battleships, and he was submitted to high temperature and considerable anxiety. He did not come under treatment till close upon a year after the disease had started, but his limits were so low as to make it practically impossible for the treatment to be continued other

than by continuous starvation; and since he was extremely thin when he came into hospital, I thought it advisable to let him have comparative comfort during the short time he was likely to remain with us. He was quite happy and cheerful until he developed coma.

Yesterday I received a letter from Dr. F. M. Allen in reply to a request to let this Society know his latest results; and, as a model of modesty and guarded prophecy, I have pleasure in reading it to you:—

“DEAR SIR,—We have had, approximately, seventy cases here. Dr. Joslin has had more than that, and the treatment has also been used rather widely in this country. Our longest experience to date is approximately two years. Naturally, we do not claim to have a cure for diabetes, but the favourable results as outlined in the preliminary papers seem fully justified up to the present. Patients of the worst type are cleared up and kept clear. They feel very much better and frequently improve considerably. By a brief departure from diet they may lose as much as they have gained in several months of treatment, but it has proved feasible for them to follow diet, and with few exceptions they have done so faithfully and consistently. I now feel no hesitation, on the basis of our experiments here and the reports of others, in affirming that this method constitutes an improvement over previous methods of treatment, but just how much can be done ultimately with the severe type of cases that we take remains for the future to show.—Very truly yours, FREDERICK M. ALLEN.”

It has taken 110 years to cover the distance between the Rollo milestone and the Allen milestone. May the distance between those on the road of the future be as great, but the time taken to reach and pass them very much less!

Dr. E. I. SPRIGGS.

After the excellent description of the experimental basis and the procedure of the fasting treatment of diabetes to which we have listened, I propose to confine myself to a brief account of our experience with the method at Duff House. The treatment has not been in use long enough to justify anything like a statistical analysis. I shall therefore pick out those cases which are most instructive both as regards the results obtained and the difficulties met with.

Fasting has been used in diabetes as a means of reducing the sugar in the urine ever since it was proposed by Naunyn. It has usually, however, been prescribed with much caution, in earlier days because

it seemed unlikely that it would do good to deprive the subject of a wasting disease of food, and in later days with the idea, shown to be mistaken by various workers, that because abstinence from food causes a moderate acidosis in health, it would aggravate the more severe acidosis of diabetes. But in mild types, and especially in the obese, fasting has long been found useful. I may here relate one such case, which was under treatment before Dr. Allen's work was known to me:—

Case I: Mild Glycosuria accompanying Hypo-acidity, Ileal Stasis and Obesity (Case 277).—The patient, a stout man, aged 41, sought relief for abdominal pain and distension of three years' standing, with nausea and giddiness for the last year. He was under treatment for ten weeks. On examination, the gastric juice was very poor in both acid and pepsin, the ileum, cæcum and appendix were adherent and there was marked ileal stasis. The blood-pressure was 175 mm. Hg. The urine was found to contain 6 gm. to 12 gm. a day of sugar but no aceto-acetic acid or acetone. The blood contained 0.17 per cent. of sugar. On a diabetic diet, continued for six days, the sugar varied from 5 gm. to 2 gm. Aceto-acetic acid and acetone appeared in the urine. Such a diet was undesirable from the point of view of the gastro-intestinal symptoms, and was not well borne. He was therefore advised to fast, with the object of getting rid of the sugar more quickly. After one day sugar was absent. For the next six days he took a diabetic diet of 1,600 calories. He was then tested with the following foods in turn: Bread, potato, oatmeal, milk, pear, apple, banana, grapes, orange, and puddings made with flour, 40 gm. a day of carbohydrate being given in each case. The urine remained free from sugar, but there was still aceto-acetic acid and acetone, though never enough to estimate. The diet was increased until he was taking 112 gm. of carbohydrate a day, of which 46 gm. was in bread, 46 gm. in puddings, 15 gm. in stewed fruit and 5 gm. in milk. With this the acid bodies disappeared from the urine, which remained sugar-free.

After reading Dr. Allen's writings and becoming convinced that not only glycosuria but also acidosis was benefited by longer periods of fasting, we began to make trial of the method. The fasting part of it is simple enough. The gradual increase of the food afterwards is not so easy, at all events in the more severe cases. Accurate diet lists and close daily supervision are required. We found that the figures given by Dr. Allen for the carbohydrate content of the foodstuffs did not agree with those obtained in our laboratories for foods as served at table. No doubt the methods of preparation differ in different countries. My colleague, Dr. Pickering, constructed graduated tables of diet, therefore, based mainly upon the analyses of our own cooked foods, and these

tables we hope to publish, as I believe they would be of use to the profession.

I will now describe five cases of those we have treated by this method, selected as representing different ages and varying degrees of severity. In all, the sugar in the urine was estimated gravimetrically, and that in the blood by Bang's method as described by S. W. Cole. With Bang's method the normal blood sugar is about 0.11 to 0.14 per cent. For β -oxybutyric acid Kennaway's method was used, and for aceto-acetic acid and acetone the Scott-Wilson method. In some cases Haurtley's method was used for aceto-acetic acid alone. Ammonia was determined by Malfatti's method.

Case II: Mild Diabetes (Case 331).—The patient, a man, aged 67, who had done much hard and responsible work in different parts of the globe, had not felt so well for the last eight years. Loss of flesh and polyuria were noticed six months, and glycosuria three months ago. He was under treatment for six and a half weeks. On admission he complained of feeling dizzy and squeamish and of pains and stiffness in the thighs. The arteries were moderately thickened, the blood-pressure 130 mm. Hg. Moist râles were heard at the base of each lung. The urine contained 49 grm. of sugar and a trace of aceto-acetic acid. The blood contained 0.24 per cent. of sugar. On a diet containing 64 grm. of carbohydrate the sugar sank to 10 grm. With a further reduction of carbohydrate to 25 grm., less than a gramme of sugar persisted in the urine, and the blood sugar was now reduced to 0.14 per cent.—that is, to within normal limits. On fasting the urine became free from sugar at once. Feeding was begun after two days, the amount of carbohydrate, protein, and fat being gradually increased until a total of 3,300 calories, containing 88 grm. of carbohydrate, 123 grm. of protein and 261 grm. of fat, was taken. The urine remained sugar-free throughout, except that a trace appeared when the carbohydrate exceeded the above quantity. Aceto-acetic acid and acetone were absent and the blood sugar remained normal. All the symptoms were gone except the stiffness of the thighs, which, with suitable massage, improved greatly. The weight fell nearly 3½ lb. The patient would probably have done well without fasting according to earlier standards, but whereas formerly, if sugar appeared in the urine he would have had to remain on a strict diet for a considerable time, he will now fast a day and will then be able very soon to take again a diet containing a fair quantity of carbohydrate. In other words, he will be able to keep sugar-free with greater precision and comfort.

Case III: Mild Diabetes in an Aged Man with Hypertrophied Heart and High Blood-pressure (Case 289).—The patient was aged 79. He had lived a vigorous open-air but hard life, but had not restricted himself in any way as regards alcohol and tobacco. Thirteen years ago thirst came on rapidly,

followed soon by aching and cramps in the limbs, which have persisted. He was under treatment for six and a half weeks. On examination, he was stout, with thick arteries and an enlarged heart. The blood-pressure varied from 184 mm. Hg to 225 mm. Hg. The urine contained albumin varying from 0.5 to 1.5 parts per thousand. There were traces only of aceto-acetic acid and

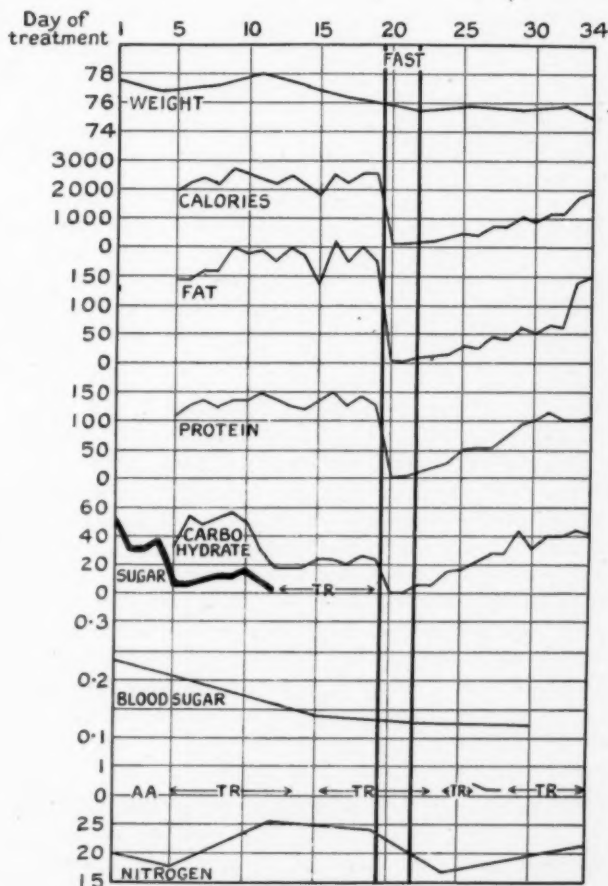


CHART I (Case 381).

Analyses by A. B. Weir, B.Sc.

acetone. The sugar ranged from 20 grm. to 40 grm.; on a strict diet it sank in ten days to 8 grm. We hesitated at first to deprive this patient of food on account of his age, and, as a tentative measure, kept him in bed one day and

allowed him two eggs only. On this day the sugar fell to 2 gm. The next three days he took a strict diet, and passed 3 gm. to 6 gm. a day. He then fasted two days, on the first of which there was a trace of sugar, on the second none. He was now very hungry, and we gave him a diet of 1,600 calories—that is, more food than the routine prescribes—with the result that a trace of sugar was present on three of the next five days, and on the other two an estimable quantity. He therefore fasted two more days, becoming sugar-free again and remaining so, while the diet was gradually increased to 1,800 calories, including 40 gm. carbohydrate in bread. A trace of sugar then appeared for two days, but after that the urine became sugar-free without further fasting, and remained so during tests with bread, potato, oatmeal, and milk. The albumin diminished, and for the last week of his stay was below five parts per thousand.

Case IV: Diabetes in a Young Man (Case 301).—The patient, aged 25, was wounded by shrapnel on active service five months before admission. During convalescence a dry mouth and thirst came on rapidly, and the urine was found to contain 10 per cent. of sugar. Weakness and aching of the legs followed. On admission, he was rather thin with a clear skin. The urine contained 174 gm. of sugar, 0.8 gm. of aceto-acetic acid and 0.1 gm. of acetone; 6 per cent. of the total nitrogen was in the form of ammonia. Two drachms of sodium bicarbonate rendered the urine alkaline in twenty-four hours. With gradual restriction of carbohydrate the sugar sank in three weeks to 20 gm. in a day, the aceto-acetic acid increasing a little—namely, to 1.6 gm. A fast of one day did not clear the urine, and after an interval he fasted three days, the urine becoming sugar-free on the second. The diet was then gradually increased and the tolerance tested for measured quantities of bread, potato, oatmeal, rice, apple, banana. From these 98 per cent. to 100 per cent. of the carbohydrate was assimilated. He was able to take 70 gm. of carbohydrate. On several occasions when the amount of carbohydrate given touched the limit of tolerance, a little sugar appeared in the urine, but was always abolished by reducing the amount in the food. The patient went away sugar-free, the symptoms having vanished and the weight increased by 10½ lb. He and his medical adviser were provided with a scheme of diet based on the analytical results obtained.

The three preceding cases are examples of different types. They include patients of ages ranging from 25 to 79. They show that fasting for three days and under-feeding for several days after did not give rise to any harmful or distressing symptoms, and led to much improvement in the feeling and condition of the patients. The urine became free from sugar and acid bodies without delay, and remained so on a more liberal diet than that taken before treatment.

I may add that regular fasts have been used with excellent results under my direction in diabetes complicated by phthisis.

In the two following cases, more severe in type, the treatment, though well borne by the patients, did not give such good results:—

Case V: Moderately severe Diabetes (Case 337).—The patient, aged 33, had lived in India for several years. While on active service in the Mesopotamian

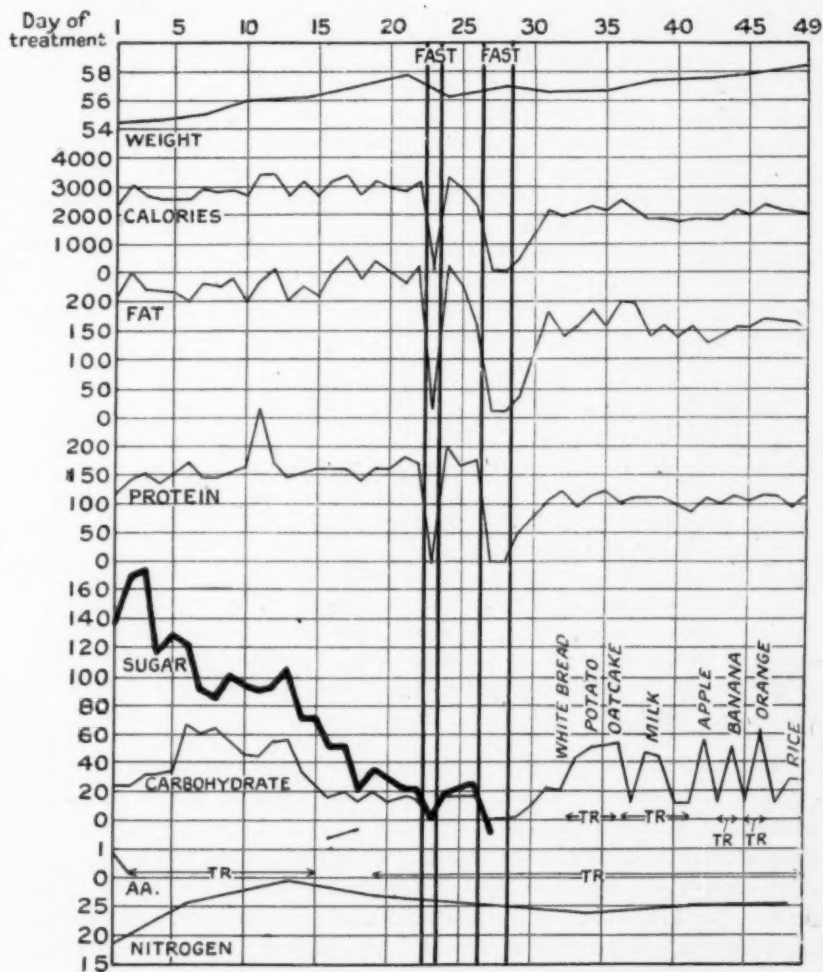


CHART II (Case 301).

Analyses by A. B. Weir, B.Sc.

campaign, thirst, polyuria, and blurred vision came on suddenly, and the weight soon fell away. On examination three months later he was moderately thin, the pulse 112, blood-pressure 164 mm. Hg. On a diet similar to that taken before admission, which included about 80 gm. carbohydrate, the urine contained 83 gm. sugar, 2.2 gm. of β -oxybutyric acid, and 0.6 gm. of aceto-acetic acid and acetone; 5 per cent. of the total urinary nitrogen was in the form of ammonia. The blood contained 0.19 per cent. of sugar. With reduction of carbohydrate to 30 gm. a day (in greens and milk), the protein in the food being kept constant, the blood sugar fell to 0.14 per cent., and that in the urine to 14 gm. a day, with 1 gm. of oxybutyric acid and 0.5 gm. of aceto-acetic acid and acetone. He then fasted two days. There was a trace of sugar on the first day, none on the second. The blood sugar sank to 0.10 per cent. The diet was gradually raised in nine days to 2,000 calories, including 70 gm. carbohydrate, without glycosuria. The acid bodies almost disappeared also, the total of the three being only 0.2 gm. On giving more food, however, sugar appeared. A day's fast got rid of it, but it was not found possible this time to increase the food to the same extent as before, for sugar appeared and rose steadily even when no carbohydrate was taken except that in green. Thinking that we might be giving some unsuitable form of protein or too much fat, we gave all the protein on one day as fish, and on another as eggs, and on a third day gave less fat, but the sugar continued to rise. Ten days after the last fast the patient fasted again, this time for three days, the urine being sugar-free for the last two of them. The blood sugar was 0.12 per cent. Food was gradually added until, nineteen days after the last fasting day, a trace of sugar was found. At this time 2,200 calories with 33 gm. carbohydrate was being taken. The blood sugar was 0.13 per cent., that is, within normal limits. The urine was again cleared by a two days' fast, the blood sugar falling to 0.10 per cent. The weight had now fallen 10 lb. in eight weeks, and the patient was desirous that we should spare him the long period of under-nutrition and increase the food more quickly. He was then treated on former lines—i.e., on a diabetic diet of sufficient heat value. The weight rose 11 lb., and the patient's sense of well-being and his energy rose with it, but sugar was present.

The chart shows well how glycosuria was postponed by making the addition of food very gradual. After the third fast it is seen that the increase of all the foodstuffs was much slower, but in each period of increasing food an estimable quantity of sugar occurred when the heat value reached the neighbourhood of 3,000 calories, which was forty-eight calories per kilogram. With 2,000 calories, giving thirty-two calories per kilogram, glycosuria was absent. The caloric value of the food—that is, the amount of protein and fat—appeared to have more effect in causing glycosuria than the amount of carbohydrate. With the reduction in carbohydrate before the fasting the blood sugar fell to within normal

limits, and the subsequent appearances of sugar in the urine were not accompanied by any obvious increase of that in the blood. But with the rise of urinary sugar at the end of the chart, the blood sugar also

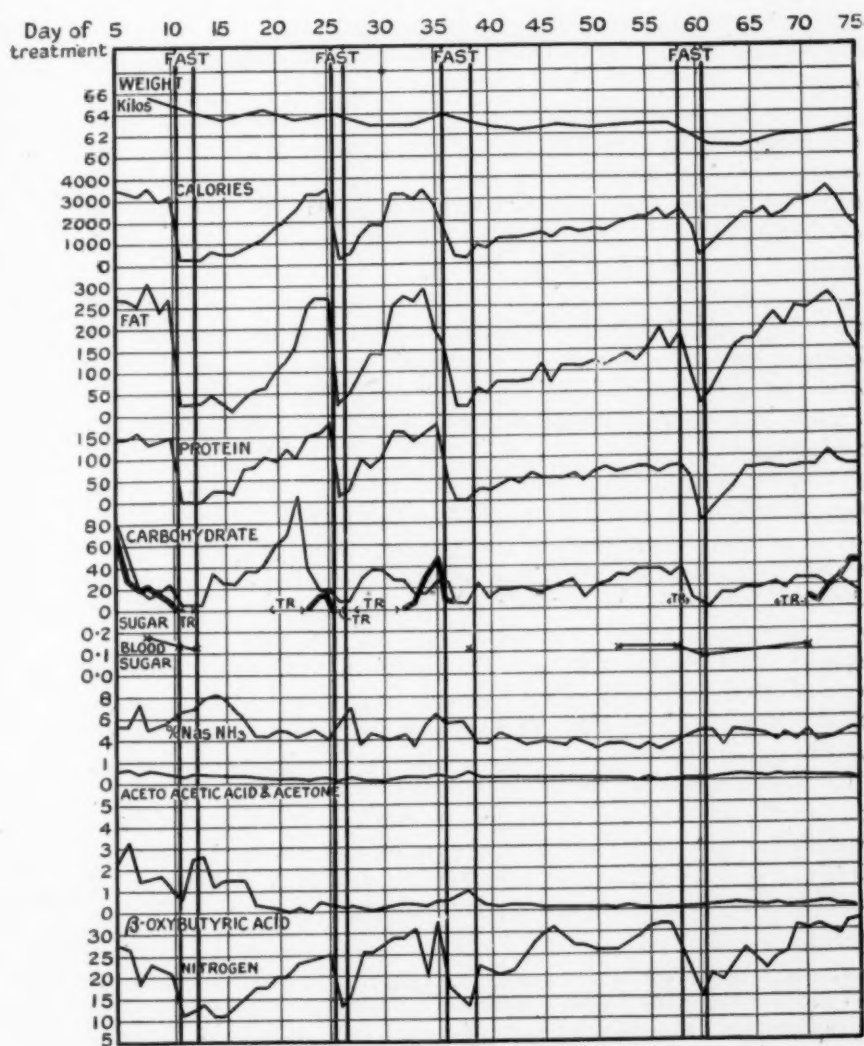


CHART III (Case 337).

Analyses by A. B. Weir, B.Sc.

rose. Foster suggests that glycosuria may be more difficult to control in patients without marked hyperglycemia. As regards acidosis, the chart shows that the first fast temporarily increased the oxybutyric acid, aceto-acetic acid and acetone, and ammonia, but this effect was less with each succeeding fast. The fall of oxybutyric acid, as carbohydrate food increased after the first fast, is well shown, the amount dropping nearly to zero in about ten days.

On reviewing the treatment it is possible that this patient would have done better if, after the first fast, a weekly fast of a single day had been given to re-establish tolerance for food, or if less fat had been given throughout. As regards the tolerance for carbohydrate, that appeared to be lower after the later fasts than at the beginning. It got worse and not better.

It is premature to draw any conclusions adverse to the treatment from this case, for in some respects Dr. Allen's instructions were not rigorously followed. I bring the case before you in some detail because it was carefully observed and because it illustrates the advantage, the difficulty and the disadvantage of the method in a moderately acute and resistant case of diabetes.

The advantage is immediate and obvious. The urine is cleared of sugar to the satisfaction of the physician and the patient. The difficulty lies in keeping the increase of protein and carbohydrate and fat a gradual one, and at the same time resisting the natural desire of the patient to get enough to eat. The disadvantage in this case was that when the patient was on a maximum diet of, say, 2,200 calories, his capacity for work and ordinary life was greatly diminished. He did not feel fit enough to drive his car even short distances for pleasure. On a fuller strict diet, although passing some sugar, he can drive, and works a couple of hours at a book he is writing, with ease and enjoyment.

Experience may show that a man lives longer if he is kept sugar-free, even at the expense of his general nutrition. But until we have evidence on that point I am inclined to think that in treating a chronic disease it is of importance to maintain the patient in such a state of nutrition that he can work and can enjoy himself.

Case VI: Severe Diabetes (Case 338).—The patient, aged 24, was on active service in France when he caught a bad cold followed by broncho-pneumonia, and was invalided home. After a month he was better but felt run down, and on examination the urine was found to contain 9 per cent. of sugar. He was kept for a time on a strict diet, and then fasted four days. The urine became sugar-free and the diet was gradually enlarged. On admission he was drowsy and the breath smelt strongly of acetone. On a strict diet of 3,000 calories the

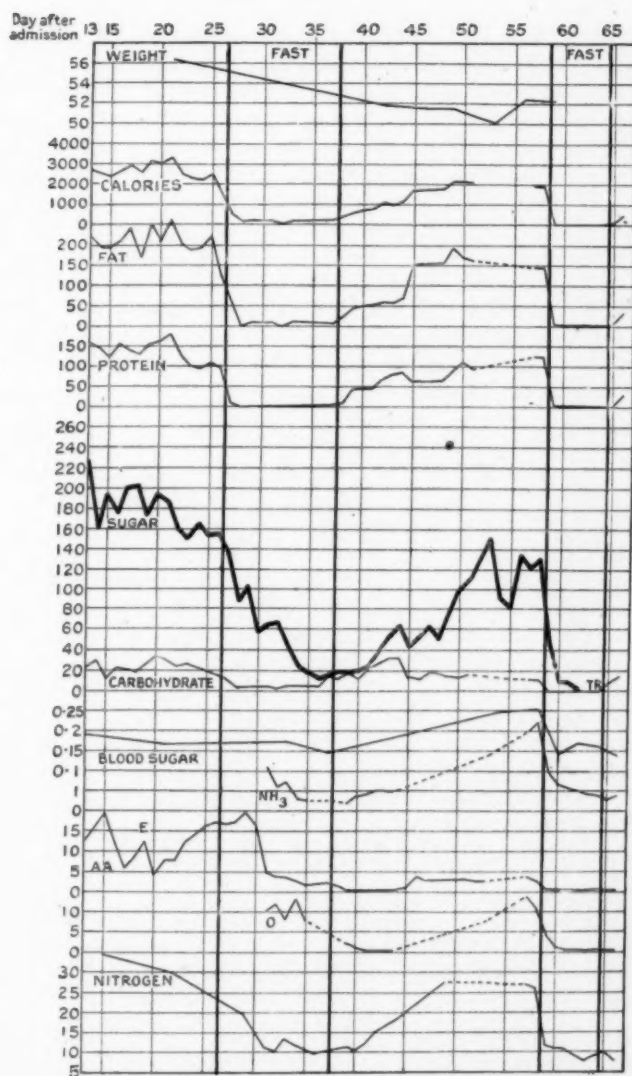


CHART IV (Case 398).

Analyses by A. B. Weir, B.Sc.

urine contained 176 grm. of sugar, 9 grm. of aceto-acetic acid and acetone and 38 grm. of nitrogen. The blood contained 0.21 per cent. of sugar. The patient's condition appeared serious, and sodium bicarbonate was given in milk up to 35 grm. (1½ oz.) a day, which made the urine alkaline. A week after admission the temperature rose and signs of broncho-pneumonia developed. The acidosis became still more severe, aceto-acetic acid and acetone reaching 20 grm. in the day. The urine became acid again, but was made alkaline by increasing the bicarbonate to 42 grm. (1½ oz.) a day. The pneumonic signs disappeared, and in a little over two weeks after admission he was feeling well. The aceto-acetic acid and acetone had fallen to 5 grm. a day. He was still taking 35 grm. to 42 grm. of bicarbonate of soda daily. The blood sugar was now 0.17 per cent. A week later a fast was begun. It was not complete, in that for the first five days 16 oz. a day of milk was drunk, as the bicarbonate was given in milk. The fast was continued for ten days, for the last five days of which no milk was taken, but at the end of that time the urine was not free from sugar. The chart shows the gradual fall from 137 grm. to 14 grm. a day. The blood sugar came down to 0.14 per cent., that is, to within normal limits. The diacetic acid and acetone were reduced from 15 grm. to 3 grm. a day. Corresponding with this the bicarbonate was cut down to 7 grm. (½ oz.) a day, the urine remaining alkaline. The acidosis was so much lessened that at the end of the fast no bicarbonate was needed for a fortnight, and then only half the quantity which had been needed before. During the latter days of the fast the patient was extremely hungry and dreamed of food, but felt nevertheless better and clearer than he had for a long time. On gradually increasing the food without any carbohydrate except in greens the blood sugar rose again to 0.21 per cent.; the glycosuria increased, but not to its former level. The acid bodies were in much smaller quantity than they had previously been on a strict diet. The patient was up. He felt weak but was cheerful. After an interval of nearly a month the patient undertook a second fast, which was continued for six days. This time the sugar sank to a low figure on the second day and on the fifth day there was none. A trace appeared on the sixth day, but the fast was nevertheless stopped, and sugar was absent on the succeeding days. The patient is still under treatment, and is now taking 1,860 calories, furnished by 99 grm. of protein, 42 of carbohydrate, and 138 of fat.

The chart shows that the percentage of blood sugar and the amount of ammonia in the urine both became normal. The oxybutyric acid, aceto-acetic acid and acetone were also reduced rapidly to very small quantities.

I must not discuss the metabolic changes in detail to-day, but may point out that this patient was not only unable to use carbohydrate in the food, but was excreting a large quantity of sugar formed from his tissues and from the protein and perhaps the fat in the food. Fasting had the effect of enabling him for the time being to burn all, or nearly

all, of the sugar produced from his tissues. Further, as less sugar was passed out afterwards it had improved his capacity for burning sugar made out of protein or fat in the food, and greatly reduced his acidosis. How far the benefit will reach remains to be seen. It is certain, however, that in a case of this severity such a good effect on the disordered metabolism cannot be attained by any other means at our disposal at the present time.

It is clear, therefore, that a fast offers the quickest means of restoring, to a greater or less degree, the power of assimilating the sugar which is made in the body from the protein, and possibly the fat, of the tissues or the food. It must not be forgotten, however, that the power to use sugar made in the body may be restored in all but the most severe cases, though more slowly, by a strict adherence to a diabetic diet. This point is of such great importance, especially to those who have to go on working, that I will venture to take up your time further by giving two examples of it, both in marked cases of diabetes, one with but little acidosis and one with acidosis well established:—

Case VII: Diabetes with Mild Acidosis treated by a Modified Strict Diet (Case 134).—The patient, aged 44, gave a family history of diabetes and phthisis. He worked several years in a tropical climate and had had dysentery. Nine years ago he was near to death for some weeks with an abscess in or above the liver, either dysenteric or secondary to a gangrenous appendix. After a slow recovery he remained in this country. For the past two or three years he has done hard and responsible work. Two months before admission thirst and polyuria came on suddenly, and glycosuria was found. On admission, the urine contained 137 grm. of sugar, 0.3 grm. of aceto-acetic acid, and a little acetone. The blood contained 0.26 per cent. of sugar. In the course of three weeks the carbohydrate in the food was diminished, and after that a strict diet was taken for a week. By cutting off carbohydrate gradually an increase of the acidosis was successfully avoided. A few grammes of sugar, however, still persisted in the urine. Tests were then made to find out what carbohydrates were assimilated best. These showed very poor assimilative power except for apples. He went away at the end of two months with about 5 grm. of sugar per day in the urine. The symptoms had disappeared. There was great gain of vigour and an increase of weight of 5½ lb. He was advised to keep to a strict diet, with an apple three times a week and two vegetable days a week. He did this conscientiously, with the result that the urine became sugar-free two months later and remained so. The blood sugar on departure was 0.22 per cent. A year after his first admission he returned to be "chemically overhauled." In the interval he had been working continuously. The urine was free from sugar. Aceto-acetic acid and acetone were present, but not enough to estimate. The blood sugar had become normal, 0.11 per

cent. On adding small quantities of starches no sugar appeared, and in the course of three weeks the carbohydrate was increased until a diet containing 81 gm. carbohydrate, 96 gm. of protein, and 180 gm. of fat was taken, yielding 2,400 calories. On this there was no sugar or acetone, and only a trace of aceto-acetic acid.

Case VIII: Diabetes with marked Acidosis treated by a Modified Strict Diet (Case 151).—A man, aged 57, had had cramps in the legs for eighteen months, had lost over a stone in weight, and felt ill. Polyuria was noticed and sugar discovered. On admission the blood-pressure was 180, the liver enlarged. The urine contained 172 gm. of sugar, 5 gm. of aceto-acetic acid, and 0.2 gm. of acetone. The blood contained 0.37 per cent. of sugar. The patient bore restriction of carbohydrate badly at first, but became accustomed to it later. The sugar did not fall, however, below 60 gm. to 100 gm. a day after three weeks on a strict diet. The aceto-acetic acid, though rising at first, settled to a level of about 2 gm. a day. Tests with various carbohydrates showed that 76 per cent. of the sugar in milk was assimilated, rather less from apples, and very little from the other forms of starch and sugar tried. After two months the symptoms were almost gone, the general health much improved, and the weight heavier. There was still, however, 50 gm. to 80 gm. of sugar a day in the urine, and 2 gm. to 3 gm. of aceto-acetic acid. The blood sugar varied from 0.30 per cent. to 0.35 per cent. He was advised to keep three absolutely strict days a week and to take an allowance of a pint of milk and an apple on the other days. After a year's work, with careful adherence to the diet prescribed, the urine is free from sugar, diacetic acid, and acetone, and the patient in good health. Small quantities of other carbohydrates are now being added to the diet. Here is the chart showing you the type of case:—

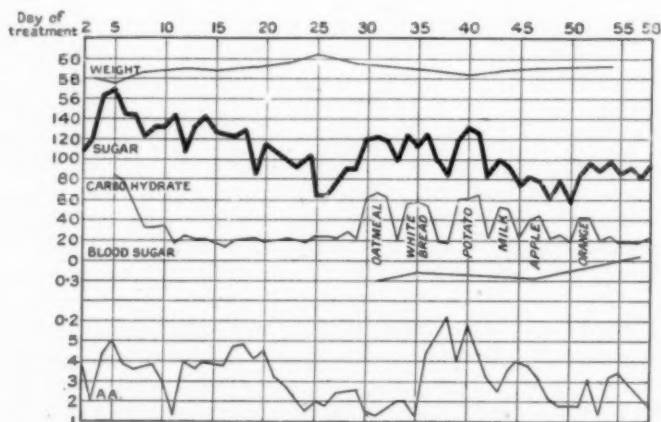


CHART V (Case 151).

Analyses by A. J. Leigh, B.Sc.

Both these patients took food of adequate caloric value, were well nourished, and able to work steadily and efficiently, the evidence of the disease becoming less all the time. I bring them before you to show you that cases which were in some respects intractable in a two months' observation did well as a result of keeping steadily to their diet, after an investigation had been made to find out what food was suitable for them.

Much time will be needed before a reasoned judgment on the fasting method can be given. At the present time I may summarize our observations as follows:—

Fasting, up to several days, was well borne by cases of mild and severe diabetes, of ages ranging from 24 to 79. The urine was made free from sugar, the blood sugar was reduced, and acidosis greatly diminished.

All the patients felt better for the fast.

The rapid abolition of sugar had an excellent mental effect; it shortened tedious treatment and enabled more time to be given to finding out what food should be taken, and in what quantity.

In most cases the food could be increased gradually, without glycosuria, until a more liberal diet was being taken than before treatment.

The gradual increase of food after the fast calls for skilled dietetic arrangements and careful daily attention; it is not so easy as an ordinary strict diabetic diet; it is useless, however, except in the mildest cases, to increase the food quickly after the fast, as sugar returns at once.

During the gradual increase of food the patient is under-nourished; in severe cases courage and endurance are called for on his part, as it may be necessary to keep him so short of food that he is inefficient.

Regular estimation of the blood sugar should be made.

In some cases of diabetes as good an effect may be attained, though much more slowly, without fasting and subnutrition, with their risk of depression and weakness, provided the patient is willing to keep permanently to the diet which has been found by careful testing to be suitable to his case.

Every case should be thoroughly investigated and treated as soon as sugar is found. With delay, it may become severer in type, needing longer fasting and greater restriction afterwards, and offering a smaller chance of arrest.

At the present time the doctor, in order to decide as to the best treatment in severe cases, must judge of the temperament of the patient. To some it is purgatory to be continually having to think about tests and

figures; these will do best with definite allowances in an otherwise rigid diet. In others the disadvantages of a half-starved condition must be balanced against the mental effect of the absence of sugar. We do not yet know whether the progress of the disease is arrested by this method in cases in which it would not be arrested by a permanent adherence to a rigid, though plentiful, diet.

For all milder cases—indeed for the majority of all cases—Dr. Allen's procedure offers great advantages. We have evidence enough that such patients, though not cured, may be freed from the signs and symptoms of their complaint. This beneficent result has been attained through the extraordinarily wide and painstaking researches of Dr. Allen on animals. None but those whose lot it has been to attempt to make researches and to describe them when made can appreciate the perseverance and the labour which have gone to the making of Dr. Allen's book and subsequent writings. I am sure we rejoice with him that he has been the means of enabling a clear step forward to be made in the treatment of a distressing complaint.

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Dr. J. H. RYFFEL.

I have collected eleven hospital cases of diabetes who have received Dr. Allen's treatment without alcohol. In six of these diacetic acid in the urine was well marked before treatment and was diminished by starvation and subsequent diet, e.g. :—

TABLE I.

Sugar, grammes <i>per diem</i> ...	350	...	54*	...	9.1*	...	5*	...	2.9
Diacetic acid <i>per diem</i> , expressed as acetone ...	1.5	...	0.75*	...	0.61*	...	0.7*	...	0.52

TABLE II.

Sugar, grammes <i>per diem</i> ...	56	...	Trace*	...	Nil*	...	Nil*	...	Nil	...	Nil
Diacetic acid <i>per diem</i> , expressed as acetone ...	0.4	...	0.22*	...	0.17*	...	0.13*	...	0.12	...	0.07

* Days of starvation.

As to the oxybutyric acid, Neubauer and also Kennaway have shown that, when the acetonuria is considerable, the amount of oxybutyric acid is roughly constant at three times as much as the diacetic acid, but when the acetonuria is slight the proportion of oxybutyric acid to diacetic acid is lower.

In four of the cases the diacetic acid excretion was slight or absent before and was slightly increased by starvation. In the remaining case there was no clear effect. The reaction to starvation was therefore similar to the normal in mild cases of acetonuria, and changed to the opposite of the normal in severe cases.

In three of the cases, all of whom had a large output of sugar before treatment, the urine did not become free from sugar. In one of these, the sugar in whose blood was 0.42 per cent., the output of sugar fell from 207 grm. to 44 grm. in one day, but rose again next day to 153 grm. After this, owing to the distress of the patient, starvation was discontinued.

In the remaining eight cases sugar disappeared from the urine. Two of these were relatively slight cases and were easily treated in this manner; one was a case with gangrene, aged 70, who did not appear to derive any benefit, and three were severe cases in whom considerable difficulty was experienced in preventing the reappearance of the sugar when food was given, so much so that it appeared a better plan to regulate the acetonuria by periodical starvation and not to aim at complete elimination of glycosuria. Another of the eight was a man, aged 44, whose leg was amputated for diabetic gangrene. Sepsis then set in. This rapidly disappeared on applying the treatment, and the sugar in the urine, which had been as much as 9 per cent., had completely disappeared by the second day of treatment. The last case was a girl, aged 9, to whom Table II refers. Sugar disappeared rapidly from the urine and the diacetic acid was markedly diminished, but there was a constant tendency for glycosuria to reappear with feeding. After three months of treatment, in which she had lost a little weight and, having grown at the same time, had become emaciated, the attempt was made to increase her weight by giving a more generous diet with restricted carbohydrate. No definite rise in weight resulted. The diacetic acid gradually increased and she finally died in coma.

Captain W. LANGDON BROWN, R.A.M.C.

At this late hour I propose only to touch on one or two points. Like others, I have always found that the fast definitely reduces the acidosis in diabetes, but I agree with Dr. Ryffel that there is the ordinary acidosis continuing which would be met with in healthy fasting individuals. What is reduced is the toxic acidosis which is productive of coma and which is due to the excess of protein and fat in the strict diabetic diet. Secondly, I agree that the temperament of the patient is a very important factor in deciding his suitability for this mode of treatment, as, indeed, it is in any restrictive treatment in this disease. The majority stand it very well once they are convinced of its advantages. Like Dr. Spriggs, I have found the days of very restricted diet more trying for the patients than the days of actual fasting.

I have never ordered a fast of more than forty-eight hours, of the value of which I am convinced. I have here two illustrative charts. The first is from the case of a little girl, aged 10. I did not like to make her fast, so I tried a series of vegetable and egg days. Each of these produced an improvement, and finally the urine was got just free from sugar; but she soon relapsed. I therefore tried a forty-eight hour fast followed by two days of vegetable and egg diet. The effect was immediate; she became free from sugar at once and has remained so for three weeks. The other chart is from a young man, aged 29. He was a fairly severe case; on a test diet containing 100 gm. of carbohydrate he excreted 112 gm., and on the various diets he always excreted more sugar than there was carbohydrate in the food, except on the potato days. Then he fasted forty-eight hours, and there was an entire change in the chart. The sugar disappeared at once and he remained free from sugar, except when the carbohydrate intake was pushed up too quickly. He was finally able to assimilate as much as 60 gm. of carbohydrate. He is feeling quite well, but sometimes passes a little sugar, which is not surprising in view of the fact that he persists in working hard in the Ministry of Munitions.

Dr. P. J. CAMMIDGE.

Fasting and a low diet have long been employed in the treatment of diabetes, but credit must be given to Allen for proving by experiments upon diabetic dogs that prolonged fasting would render them sugar-free,

and for having had the courage to apply his laboratory experience to human diabetes. The generally favourable reports from America, the cases quoted in the debate, and my own experience all suggest that the treatment of glycosuria by prolonged alimentary rest gives promising results, although it is well to bear in mind that it is of comparatively recent origin and has yet to stand the test of time, which has failed in the case of the oatmeal "cure," the potato "cure," &c. Whether the fasting treatment will ultimately fulfil present expectations depends upon the answer to two questions:—

(1) Is diabetes invariably due to defects in the internal functions of the pancreas?

(2) Is it an inherently progressive disease or simply "a weakness of a metabolic function"?

If there is a functional element in all cases of diabetes, and the pancreas is always at fault, complete rest of the weakened functions may be expected to control the symptoms and allow of repair, but if glycosuria is a progressive disease, dependent at times upon organic changes in organs other than the pancreas, complete recovery is unlikely to result from alimentary rest alone, and in some instances it may fail even to improve the condition of the patient. The dangerous increase in the acidosis, &c., observed by Stillman in some 10 per cent. of his cases while undergoing Allen's treatment points to there being limitations to the usefulness of the method. Most observers now hold that in diabetes there is a disturbance in the chemistry of the body which involves the metabolism of proteins and fats as well as of carbohydrates. This is recognized and provided for to some extent in Allen's treatment by the way in which the diet is cautiously extended after the preliminary fast and the subsequent trial of carbohydrate foods. I heartily welcome the quantitative regulation of the food supply that forms a necessary part of the procedure, for it is an important means of controlling the disease which I have been advocating for some years. The gradual addition first of carbohydrate, then of protein, and ultimately of fat, has much to recommend it, but the way in which the addition of protein is entirely governed by the absence or presence of sugar in the urine and the final fixing of the protein intake by an empirical standard are open to criticism. It is probable that more satisfactory and permanent results will be obtained if the intake of protein be governed by a determination of the nitrogenous end-products in the urine and the diet be finally arranged so that the patient is in a state of nitrogenous equilibrium. With any system of dieting it is most important that the patient should

be educated so that he can carry out his subsequent diet scheme intelligently, and with Allen's plan education of the patient is one of the most essential factors in success. It is therefore necessary that the physician should be as well acquainted with the properties and dosage of foods as he is with drugs and be able to prescribe and combine them with the same certainty. The effects of cooking and other culinary manipulations on the carbohydrate, protein, fat, and caloric value of various food materials must be understood, and he should be able to change the diet as circumstances and season demand if he is to be in a position to instruct his patient and save him from the monotony which is the cause of failure in so many systems of dieting. When using the published tables for working out a diet it should be borne in mind that most of them are based upon analyses of the raw foods, and that while the inedible waste is included in some figures it is excluded in others. It is much the best to work out the diet for foods as they are served at table, as I have done in my book on glycosuria. Although it is yet too early to grow unduly enthusiastic and to prophesy that Allen's method will replace all other forms of treatment for diabetes, there seems to be no doubt that, intelligently carried out, it removes the glycosuria and acidosis quickly and surely in the majority of instances, and that it affords relief in many cases that do not respond to other methods of treatment.

CORRIGENDUM.

The note from Dr. Panoupoulos (of Athens) on a "New Treatment of Tuberculosis" (p. 61) should have appeared in the Proceedings of the Electro-Therapeutical Section.

